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This report assesses the risk of exposure to mercury. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in August of 1980.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of mercury in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to mercury for various subpopulations.

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AN EXPOSURE AND RISK ASSESSMENT FOR MERCURY

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OFFICE OF WATER REGULATIONS AND STANDARDS
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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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Office of Water Regulations and Standards

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The materials balance for mercury (Chapter III) was adapted from a draft report by Versar, Inc., produced under contract 68-01-3852 to the Monitoring and Data Support Division, Office of Water Regulations and Standards, U.S. EPA. Justine Alchowiak was the task manager for Versar, Inc.

CHAPTER I.

EXECUTIVE SUMMARY

The Monitoring and Data Support Division, Office of Water Regulations and Standards, the U.S. Environmental Protection Agency, is conducting an ongoing program to identify the sources of, and evaluate the exposure to, 129 priority pollutants. This report assesses the exposure to and risk associated with mercury.

RISK CONSIDERATIONS

Humans

Mercury occurs naturally in many rock strata and soils at trace levels, and as a consequence, virtually all surface water and ground water contain very low levels of mercury (generally less than 1 ug/l). Because mercury is relatively volatile, atmospheric contamination can occur from natural and industrial sources. Human activities are clearly associated with increases in regional background levels, even considering natural sources. For instance, ambient air levels in urban areas appear to be about three times those in rural areas. Also, mercury levels in all media are higher in the immediate vicinity of large sources such as copper smelters, chlor-alkali plants and steam power plants.

Consideration of the sources and fate of mercury in the environment suggests a number of potential exposure routes for humans. Human intake of total mercury from food in the U.S. typically ranges from 5-15 ug/day and inhalation exposure in general ranges from 0.1-0.6 ug/day. Average ingestion of total mercury from drinking water is less than 1 ug/day. Highest exposures are very likely attained by dentists (60-6000 ug/day, by inhalation) and a small subpopulation who derive most of their diet from fish.

Mercury compounds can be absorbed through the gastrointestinal tract, the respiratory tract, and through the skin. The absorption rate and toxicity, however, vary with the route and the form of mercury and thus exposures are not additive. Of special significance is methylmercury, which is 80% to 100% absorbed from the gastrointestinal tract and has a longer half-life (70 days) than other forms of mercury.

The critical organ systems in man are the central nervous system and the kidneys. Mercury poses a particular hazard to the developing embryo. Elemental and methylmercury readily cross the placental barrier, inducing a variety of developmental anomalies and fetal death. A wide variety of malformations has been produced in laboratory animals exposed to mercury *in utero* at doses as low as 2.5 mg/kg maternal body weight. The human fetus, and specifically the fetal nervous system, appears to be particularly susceptible to methylmercury, as indicated by the mercury poisoning episodes at Minamata and Niigata. (There is no evidence to suggest that mercury compounds are carcinogenic, although methylmercury has been implicated as a mutagen.)

The primary route of human exposure to mercury appears to be through eating fish or shellfish. The World Health Organization (WHO) has recommended that weekly intake be limited to less than 200 ug of methylmercury and less than 300 ug total mercury. Similarly, a critical daily intake of 30 ug mercury, which corresponds to a mercury blood level of 20 ng/g, has been estimated by some researchers to be a safe intake for an average 70-kg man. However, there is some disagreement as to what constitutes a "safe intake." A safe fetal exposure has not been established.

An estimated 0.1% to 0.2% of the population receives more than the estimated "acceptable daily intake" (0.43 ug/kg/day) of methylmercury for neurological disturbances due to consumption of seafood containing mercury. However, the sensitivity to mercury varies within this subpopulation. In addition, a very small population (estimated to be less than 0.01% of the U.S. population) may receive exposures of greater than 1.4 ug/kg/day through seafood consumption.

With the raising of the FDA action level to 1.0 ug/g mercury in fish, a consumption of 30 g/fish/day containing the allowable level of mercury will result in exposure equaling the estimated "acceptable daily intake." Though this consumption level is probably very common in the United States at this time, mercury levels in seafood are generally below 1.0 ug/g.

An examination of the areas of the country in which fisheries have been restricted due to mercury contamination showed that in many cases the sources are unknown. Natural sources, abandoned chlor-alkali plants, and an abandoned gold mine appear to be the sources of contamination when they could be identified.

Both fetuses and children may be at risk due to large exposures to mercury; however, the risk potential cannot be quantified at the present time.

The accidental exposure of consumers to mercury through the use of mercury-containing products does represent a risk, albeit unquantifiable, to a very small subpopulation. Among these is the ingestion of small mercury batteries by children, since these batteries are becoming more widely used in the home. Degradation of the casing may expose the child to a potentially lethal dose.

Biota

Monitoring data obtained in 1979 indicate that mercury levels in surface waters at a number of locations are above the laboratory threshold for sublethal effects on the "most sensitive" aquatic species. However, LC_{50} values for "most sensitive" species are generally more than

10 times the average river basin concentrations. Fish-eating wildlife living near contaminated waters may be at significant risk due to bioaccumulation of mercury in fish.

The lowest concentration at which effects have been observed in aquatic organisms is <0.01 ug/l methylmercury, a chronic effects value for Daphnia magna. Growth was inhibited in rainbow trout at methylmercury concentrations as low as 0.04 ug/l.

The acute toxicity of mercury to fish is generally in the ug/l range, with the organic compounds, especially methylmercury, more toxic than the inorganic compounds. Mercury toxicity to invertebrates varies; aquatic insects appear to be relatively tolerant.

Selenium appears to mitigate the adverse effects of mercury on aquatic organisms as it does for humans. However, the mechanism is not well understood.

Aquatic organisms may be commonly exposed to mean total mercury levels of greater than 0.5 ug/l in the North Atlantic, Ohio River, South Central Lower Mississippi River, Pacific Northwest and California River Basins. In general, levels of mercury appear to be decreasing with time, and maximum levels of >10 ug/l occur only rarely. In addition, no fish kills attributed to mercury have been reported.

Thus, aquatic organisms may be at risk due to mercury exposure in some locations. However, methylmercury, which is the more toxic form in the laboratory, is found only at very low levels in natural waters. The risk to aquatic organisms cannot be quantified with the available data. However, the lack of evidence of fish kills associated with mercury suggests that risk due to mercury is low.

Studies of the effects of mercury on terrestrial organisms have been limited. Dietary concentrations of 3 mg/kg methylmercuric chloride produced adverse reproductive effects in mallards and black ducks. Oral doses of 13 mg/kg and 60 mg/kg were lethal to goshawks and ducklings, respectively.

Most terrestrial organisms do not appear to be at risk, except perhaps in the vicinity of anthropogenic sources. Elevated mercury residues have been found in plant and animal specimens collected near chlor-alkali plants, although no toxic responses have been reported. Piscivorous mammals and birds may be exposed to more mercury than other animals due to their position in the food chain.

MATERIALS BALANCE

Production

Mercury production in the United States in 1976 totaled 2,428 kkg. Of this amount, about 800 kkg were mined domestically in California and Nevada, secondary production from mining and extraction of other ores amounted to about 120 kkg, and the remainder (about 1,500 kkg) was imported.

Uses

The pattern of mercury use in the United States has changed and the amounts consumed have been declining slowly over the last 15 years. This is largely the result of increasing concern over the toxic and persistent nature of elemental mercury and its compounds.

Mercury consumption in the manufacture of electrical apparatus has, however, been increasing, possibly because of the increased use of mercury cells in smoke alarm devices. In addition to mercury cells, mercury is used in other batteries, lamps, switches, and rectifiers. This industry category consumed about 1,000 kkg of mercury in 1976.

The second largest user of mercury is the mercury-cell component of the chlor-alkali industry, which uses mercury as a flowing cathode for the electrolytic preparation of chlorine and caustic soda. Approximately 550 kkg of mercury were consumed in this way in 1976.

Other commercial applications for mercury compounds include use as a mildewcide or preservative in paint (270 kkg), as a constituent of pharmaceuticals (2 kkg), and as a catalyst in the synthesis of vinyl chloride and vat dyes (44 kkg). Elemental mercury is used in the manufacture of industrial instruments (175 kkg). About 70 kkg of mercury are consumed as an amalgam in dental work.

Releases

Recognized natural and manmade sources are estimated to release 3,700 to 3,900 kkg of mercury to the environment each year. By far the largest initial receptors of this release are the air and land compartments (1,662 kkg and 1,807 kkg, respectively). Approximately 300 kkg are released to the aquatic environment.

Mercury can be detected in the earth in nearly all crustal deposits. Consequently, outgassing of the earth's crust and runoff from natural erosion together contribute about 1,200 kkg in releases each year, or 31% of the known releases. The releases to the atmosphere from outgassing (~1,000 kkg) account for about 60% of all known releases to this medium, those from runoff (~200 kkg) constitute nearly two-thirds of known releases to the aquatic environment.

Releases from anthropogenic sources are estimated to total about 2,300 kkg each year. Major anthropogenic sources to the atmosphere include paint volatilization (~200 kkg) and fossil fuel combustion (~180 kkg). Releases to land are largely attributed to disposal of electrical apparatus (~780 kkg) and wastes from chlor-alkali plants (~530 kkg).

Mercury in fertilizer, POTW sludges, discarded paints and painted items, catalysts, and industrial and control instruments account for most of the remainder of releases to the land. Sources to water include manufacture and disposal of electrical apparatus (~16 kkg), application of mercury-containing paint (~24 kkg), and dental uses (~17 kkg). About 1% of known releases go to POTWs. In addition, mercury is known to contaminate urban runoff.

Considerable uncertainty is associated with the estimates of releases from mercury-containing industrial and consumer products (primarily paint and electrical equipment). Since these two product classes consumed about one-half of the 2,400 kkg used in the United States in 1976, the inability to characterize reliably the fate of mercury in these products is troublesome.

FATE AND DISTRIBUTION ON THE ENVIRONMENT

Monitoring Data

Mercury is virtually ubiquitous in the environment though elevated levels are found consistently near anthropogenic sources and occasionally near natural sources.

Mercury levels in uncontaminated freshwater and saltwater are generally low (0.04 ug/l to 0.3 ug/l). Values of up to about 50 ug/l mercury have been reported for water in contaminated areas. Sediment levels range from ~0.05 mg/kg in unpolluted areas to over 2.0 mg/kg near industrial sources of contamination. Rocks and uncontaminated soils contain 0.02 mg/kg to 0.15 mg/kg mercury, with concentrations of up to 250 mg/kg reported for sites near natural mercury deposits.

Atmospheric mercury in remote areas is primarily in the form of a vapor and is usually in the elemental form. The ratio of mercury vapor to mercury adsorbed to particulates is quite variable in urban areas. Background concentrations range from 1 ng/m^3 to 50 ng/m^3 while urban levels vary from 2 ng/m^3 to 60 ng/m^3.

Freshwater fish usually have slightly higher mercury levels (0.05 mg/kg to 1.80 mg/kg) than do marine fish (below 0.3 mg/kg). Terrestrial biota also contain detectable levels of mercury. Trees and herbaceous growth in unpolluted areas have concentrations ranging from 0.02 mg/kg to 0.03 mg/kg, with levels up to 1.25 mg/kg in areas contaminated by anthropogenic or natural sources of mercury. Levels in birds and mammals vary depending on such parameters as species and geographical region. Feeding habits can also influence mercury accumulation in mammals and birds.

Environmental Fate

Mercury in the water column is concentrated on suspended solids and in sediments. Methylation of mercury is promoted both biologically and abiologically in low pH environments, and under slightly reducing conditions. In the atmosphere, most of the mercury (>90%) occurs as a vapor, while the remainder exists adsorbed to sub-micron particulate matter. Fallout and washout will remove nearly all of the adsorbed mercury; the vapors are more prone to wide dispersal with a mean residence time of 4 to 11 days, and eventually contribute to background concentration levels. Mercury has a great affinity for organic matter, clays, and hydrous metal oxides, and in soils remains bound, provided the pH remains neutral to alkaline. Mercury may be lost from soils by volatilization; this tendency increases as the soil organic matter and moisture content decrease.

Mercury disposed of on the ground in mine tailings, coal piles, or solid wastes is a major source of mercury to the environment. However, little evidence exists to suggest that mercury enters surface or ground waters as a result of acid mine drainage, or leaching from tailings and landfills. Clays and organic matter in soils effectively reduce the quantity of mercury leached from these systems. Soil environments favoring transportation of mercury would be low in pH and contain little clay and organic matter.

Phenylmercurials, which constitute most pesticidal forms of mercury, are easily leachable, as well as subject to loss by vaporization and surface runoff.

Mercury enters POTWs at an average concentration of 0.4 ug/l. Aerobic and anaerobic biological treatment partition more than 90% of the mercury into the sludge portion of the waste; the remainder exists adsorbed onto suspended solids. The sludge generated by POTWs is disposed of in landfills, by landspreading, or is incinerated. Sludge spread as a soil amendment is not likely to enhance the solubility or mobility of mercury. Landfill leachate analysis for mercury demonstrated concentrations no higher than 0.2 mg/l. Aqueous effluents of wastewater treatment contain mercury principally in the insoluble state. Discharge to freshwaters will most likely result in elevated sediment concentrations, and the possibility of methylation; discharge to marine waters causes solubilization and oxidation of the mercury due to dilution.

Biological Fate

Methylmercury, which is the most common form of mercury found in aquatic organisms, is rapidly accumulated and retained for long periods, with a half-life of 1000 days in some species of fish. Both ingestion and gill absorption are exposure routes for mercury, with the former appearing to play a more significant role in upper-trophic-level organisms. Once absorbed, methylmercury tends to be associated with muscle tissue -- the edible part of fish -- and liver and kidneys.

Bioconcentration levels range from one to six orders of magnitude higher than background water concentrations and biomagnification of mercury appears to occur in at least certain aquatic food chains.

Terrestrial plants generally do not accumulate mercury to very significant levels compared with aquatic biota, though conversion of phenyl and other mercury compounds to methylmercury may take place in some plants. The forms of mercury present in soil and their influence on uptake rates have not yet been determined.

CHAPTER II.

INTRODUCTION

The Office of Water Regulations and Standards, Monitoring and Data Support Division, the U.S. Environmental Protection Agency, is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated included potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This report provides a brief, but comprehensive, summary of the production, use, distribution, fate, effects, exposure, and potential risks of mercury. There are a number of problems with attempting such an analysis for this chemical. Mercury is an element commonly found in the earth's crust and releases to the atmosphere and to water from natural sources can be significant in some locations. However, the estimation of the contributions of these important sources is difficult.

In addition, the number of different forms of mercury make exposure and risk assessment complex because the absorption and toxicity varies with the route of exposure and the compound. Therefore, the form of mercury has been specified where possible.

The evaluation of risk due to mercury is also complicated by the fact that much of the toxicity data for mercury is epidemiological in nature. Thus, it is often difficult to associate observed effects with specific doses. However, the data collected at the several incidents of widespread mercury poisoning have provided invaluable insight into the effects resulting from mercury exposure.

The report is organized as follows:

> Chapter III presents a materials balance for mercury that considers quantities of the chemical consumed in various applications and produced naturally, the form and amount of pollutant released to the environment, the environmental compartment initially receiving it, and, to the degree possible, the locations and timing of releases.

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Chapter IV describes the distribution of mercury in the environment by presenting available monitoring data for various media, by considering the physicochemical and biological fate processes that transform or transport mercury, and by characterizing the major environmental pathways for releases to the environment.

Chapter V describes the available data concerning the toxicity of mercury for humans and laboratory animals and quantifies the likely level of human exposure via major known exposure routes.

Chapter VI considers toxicological effects on and exposure to biota, predominantly aquatic biota.

Chapter VII presents a range of exposure conditions for humans and other biota and compares these with the available data on effects levels from Chapters V and VI, in order to assess the risk presented by various exposures to mercury.

Appendix A contains notes concerning computations in Chapter III.

Appendix B presents an overview of the present status of restrictions on commercial and sport fisheries in the U.S. due to mercury contamination.

CHAPTER III.

MATERIALS BALANCE

A. INTRODUCTION

This chapter presents the materials balance for mercury in the contiguous United States for the year 1976. It was adapted from a draft report written by Versar, Inc.* The materials balance summarizes the principal sources, uses, and environmental releases of mercury from use categories believed to contribute more than 0.1 kkg per year to all environmental media. Releases from both anthropogenic and natural sources are considered. Potential anthropogenic sources were identified by a review of activities in which the material participates from its extraction and use in various forms to its ultimate disposal. For each major source of pollutant release, the amount of material released is estimated, the environmental compartments (air, land, water) initially receiving and transporting the material are identified, and the locations at which the pollutant loadings take place are specified to the degree possible.

Data were obtained from a large number of published and unpublished reports. The publication of greatest use in developing this materials balance was the comprehensive report on mercury and its compounds prepared by URS Research Corporation for the U.S. Environmental Protection Agency (Van Horn 1975). This URS report pertains to the early 1970s, and some of the data have been supplanted by subsequent data from actual field measurements. In these cases, the more recent data, which are thought to be more accurate, have been used. For example, URS cites the emission factors for mercury used in the chlor-alkali industry as 0.035 to air, 0.004 to water, 0.501 to land, and 0.46 to inventory and recycle. A study by Versar, Inc. (1976a), conducted during 1975 and 1976, included visits to 16 of the mercury-cell chlor-alkali plants in the continental U.S., and data developed indicate that environmental distribution factors are more accurately reflected in the following numbers: 0.033 to air, 0.001 to water, 0.005 to the product caustic, and 0.962 to land (including non-discharging brine wells, evaporation ponds, and sludge ponds, sludge pits and landfills). Similarly, screening and verification sampling data collected by the Effluent Guidelines Division of EPA were available for several industries (U.S. EPA 1979c). For aquatic discharges and discharges to POTWs, these data are assumed to be the best available and were used in the materials balance.

The year 1976 was selected for the analyses because it is the most recent year for which a complete set of data were readily available.

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*Environmental Materials Balance for Mercury. Draft report to the Monitoring and Data Support Division, Office of Water Planning and Standards, U.S. EPA, 1979.

However, data on reported and apparent mercury consumption supplied by the Department of Commerce for the period 1976-1977 indicate that mercury use patterns did not change significantly between 1976 and 1978, the latest year for which there are useful data from which to construct at least a partial materials balance. The uncertainties in the data on environmental releases far exceed any variations in the apparent and reported consumption levels. Therefore, the environmental releases reported for 1976 are probably representative of the releases for 1978 also.

Table 1 presents a breakdown of the amount of mercury discharged to the U.S. environment from recognized natural and man-made sources during 1976. Of the approximately 3700-3900 kkg of mercury identified as entering the total environment each year, 31% (~1200 kkg) is derived from natural sources. Figure 1 shows the total mercury flow in the U.S. in 1976, tracing the cycle from production sources to disposition in the environment. Figure 2 displays the same data in a schematic diagram that combines both flows and volumes in order to illustrate the relative contributions of sources to each environmental compartment.

Although the amount of mercury released by natural sources is probably relatively constant from year to year, man-made releases are probably roughly proportional to consumption and thus more variable, except that the release of mercury from some products may be delayed a number of years until the containment vessels decay. During 1976, consumption of mercury was 25% to 40% greater than in 1975, and was the largest since 1969. The increase in mercury consumption in 1976 was primarily due to an increase in electrical apparatus manufacture and, within that industrial category, to a large increase in the use of mercury cells in smoke alarm devices.

The distribution of mercury released to the environment in 1976, as shown in Table 1 and Figures 1 and 2 is summarized below:

	Mercury Release	
Environmental Receptor	Total (kkg)	%
Air	1625-1696	43
Water	308-312	8
POTWs	41	1
Solid Waste	1754-1858	48

The major recipients of the various mercury releases are the air compartment and the land. However, virtually all of the mercury that goes to land is derived from industrial operations of one kind or other (manufacturing, fossil fuel combustion, and mining and smelting operations), whereas only about 40% of emissions to air are anthropogenic, the rest being from natural, and therefore, uncontrollable

TABLE 1. PRODUCTION AND USE/RELEASES OF MERCURY IN THE UNITED STATES, 1976

Production (kkg)		
Commerce		2428
Mine and Primary Prod.[1]	797	
Secondary Production[1]	116	
Imports[1]	1515	
Fossil Fuel Combustion[17,18,22]		192
Impurity or byproduct[23,24,26,27]		251
Fertilizer[27]	190	
Copper mining[6]	41	
Other[6]	18	
Natural Sources[6]		1207
Rural Runoff and Groundwater[6]	188	
Degassing of Earth	1019	
Total Supply	4077	

Use (kkg)		
Mfg. of Mercury Containing Products		1683
Electrical Apparatus[2]	948	
Paint[2,3]	270	
Indust. Control Inst.[2]	175	
Dental Preparations[2]	69	
Catalyst Manufacture[2]	44	
Fungicides/Bactericides[4]	21	
General Laboratory Use[2]	20	
Pharmaceuticals[2]	2	
Other[2]	116	
Mgfr. of Chlorine & Caustic[2]		553
Exports[2]		17
Unaccounted for (including stockpiles)[5]		175
Commercial Use Subtotal		2428

Source	Release (kkg) Air	Water	POTW[a]	Land	
Media Totals	1662	299		1802	3763
Reprocessing of Mercury & Products[6,20,21]	7.8	0	0	0.4	
Mfg. of Mercury Containing Products[7,9,10,12,13]	0.2	0.1	0.4	11.4	
Manufacture of Chlorine & Caustic[8]	18.5	0.3	0	538.1	
Use of Mercury Containing Products					
Electrical Apparatus[16]	141	16	0	779	
Paint Applications[9]	199	26	0	47	
Industrial Control Instruments[10]	6.9	0	0	97	
Dental Preparations[11]	1.4	16.5	11.6	17.1	
General Laboratory Use[14]	2.1	-	5.1	1.6	
Catalysts[12]	0	0	0	41.3	
Agricultural Use of Fungicides & Bactericides[13]	0	3.1	0	17.9	
Pharmaceutical Applications[15]	0	0.6	1.3	0.2	
Fossil Fuel Combustion[17,18,22]	177	4.6	0	10.5	
Impurity or Byproduct					
Fertilizer Impurities[27]	-	-	-	190.5	
Copper Mining[6]	40.8	0	0	2.1	
Other[6]	10.0	4.8	0.7	2.8	
From POTW[19]	36	42		50	
Natural Sources					
Rural Runoff and Groundwater[6]	0	188	0	0	
Degassing of Earth's crust[6]	1019	0	0	0	
Exports[2]					17
Unaccounted for (including industrial stockpiles, recycled mercury, and unknown releases)					297
					4077

[a] Releases to POTW have not been added to the total releases, since these have been accounted for in POTW releases.

Note: Detailed notes explaining the derivation of numbers in this table are given in Appendix A.

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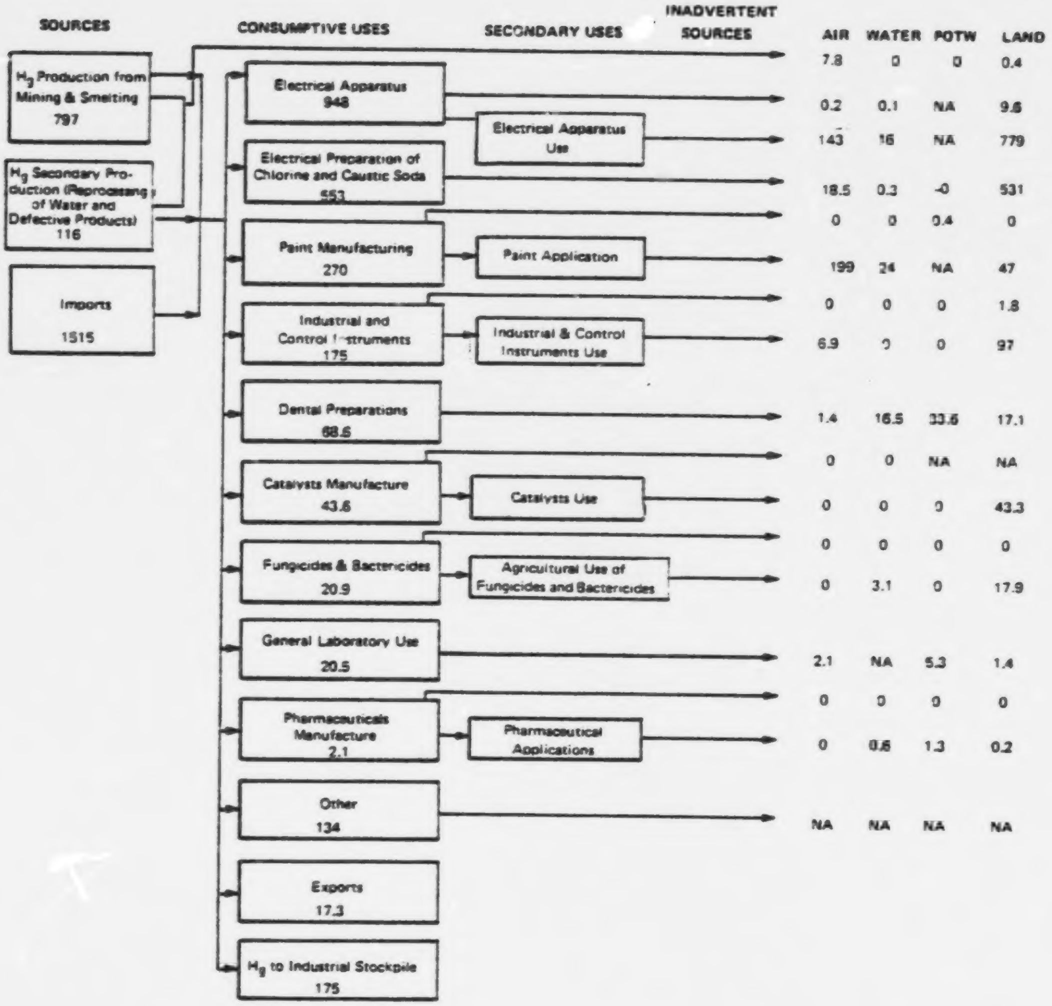

SOURCES
CONSUMPTIVE USES
SECONDARY USES
INADVERTENT SOURCES
AIR
WATER
POTW
LAND
Hg Production from Mining & Smelting 797
Hg Secondary Production (Reprocessing of Water and Defective Products) 116
Imports 1515
Electrical Apparatus 948
Electrical Apparatus Use
Electrical Preparation of Chlorine and Caustic Soda 553
Paint Manufacturing 270
Paint Application
Industrial and Control Instruments 175
Industrial & Control Instruments Use
Dental Preparations 68.6
Catalysts Manufacture 43.6
Catalysts Use
Fungicides & Bactericides 20.9
Agricultural Use of Fungicides and Bactericides
General Laboratory Use 20.5
Pharmaceuticals Manufacture 2.1
Pharmaceutical Applications
Other 134
Exports 17.3
Hg to Industrial Stockpile 175
7.8 0 0 0.4
0.2 0.1 NA 9.6
143 16 NA 779
18.5 0.3 -0 531
0 0 0.4 0
199 24 NA 47
0 0 0 1.8
6.9 0 0 97
1.4 16.5 33.6 17.1
0 0 NA NA
0 0 0 43.3
0 0 0 0
0 3.1 0 17.9
2.1 NA 5.3 1.4
0 0 0 0
0 0.6 1.3 0.2
NA NA NA NA

Note: Values are given in kkg.

FIGURE 1 FLOW DIAGRAM FOR CYCLE OF MERCURY PRODUCTION, CONSUMPTION AND DISPOSITION IN THE UNITED STATES

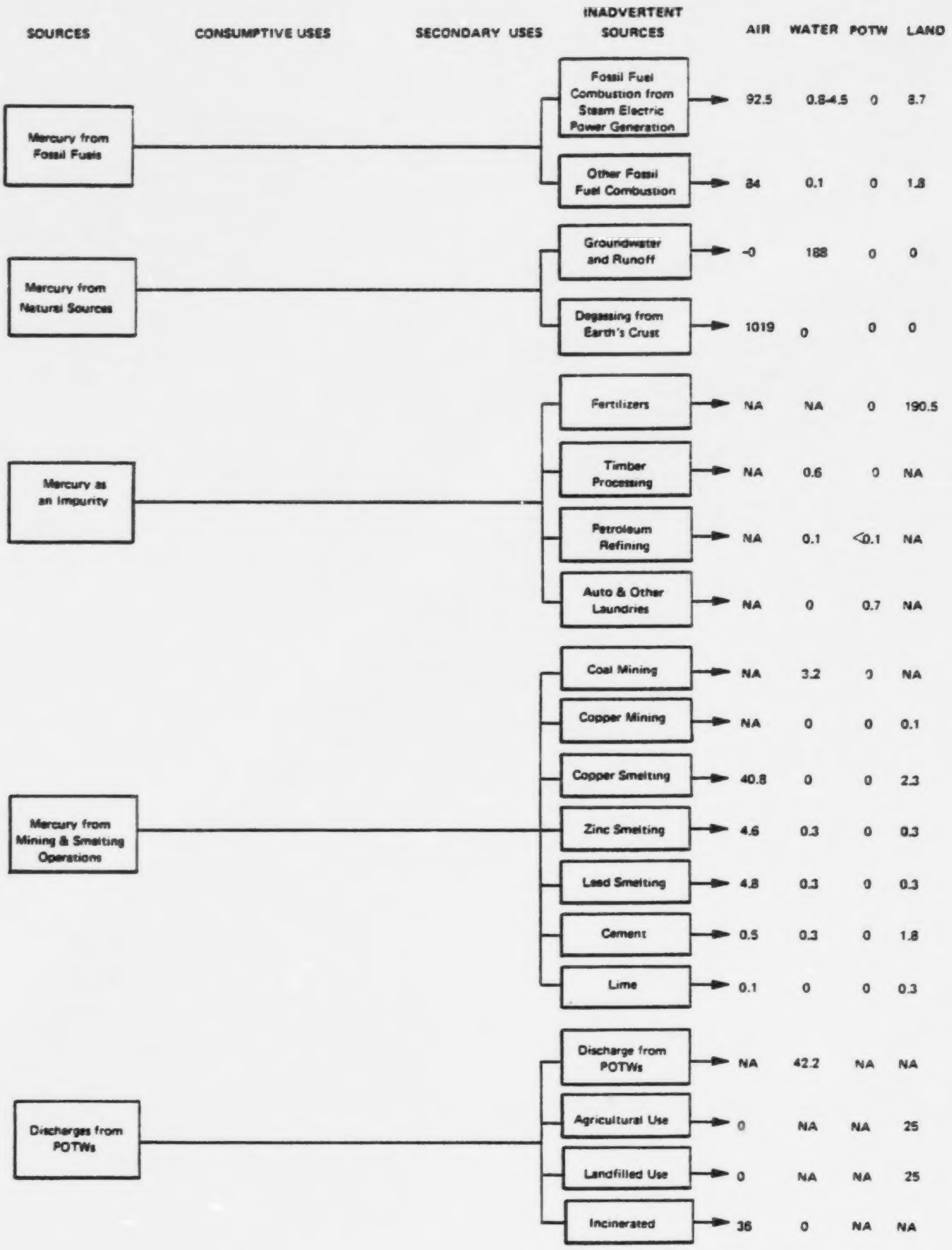
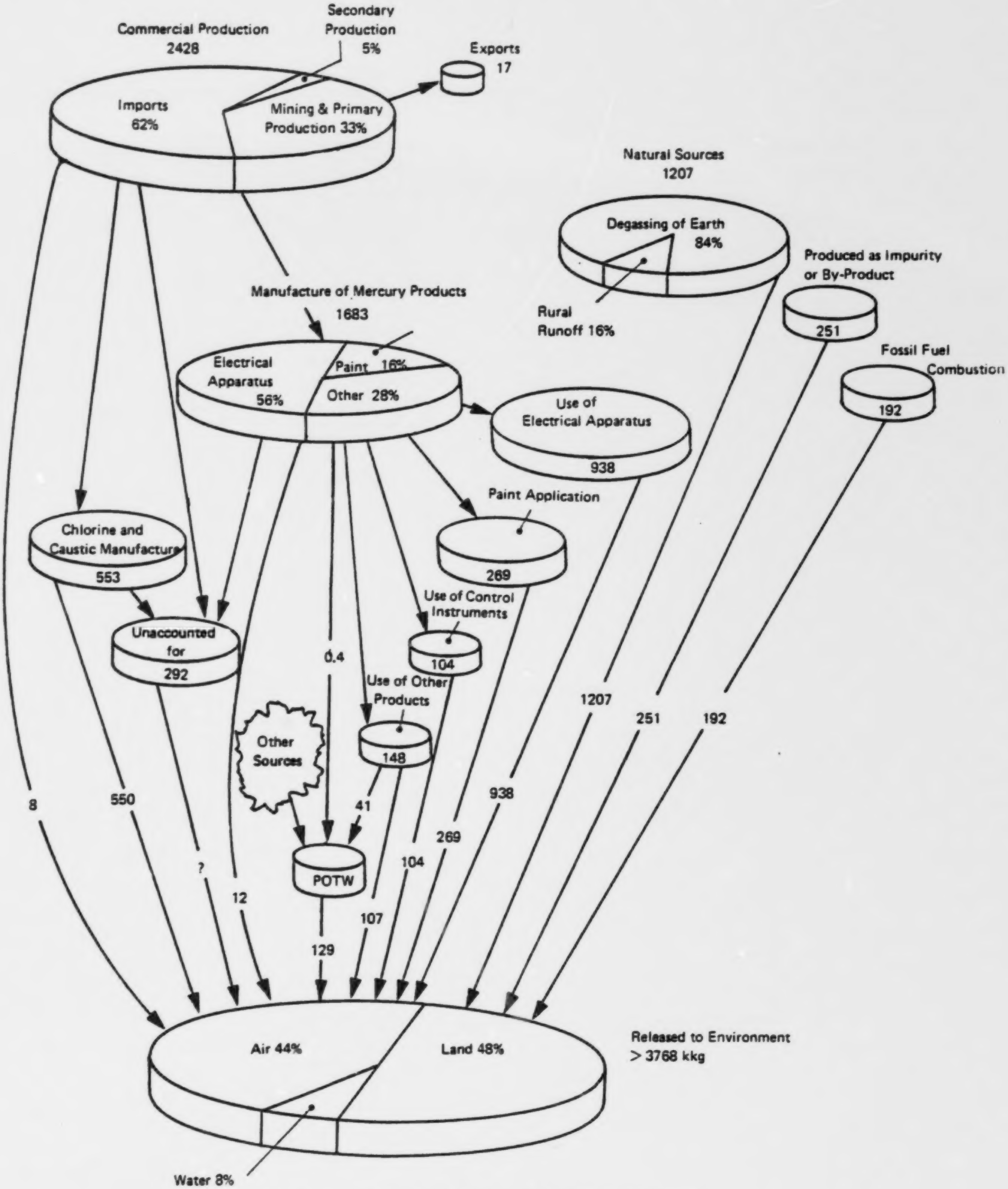


FIGURE 1 CONTINUED



Note: All amounts are in kkg. FIGURE 2 MATERIALS BALANCE OF MERCURY—1976 sources. In addition to these sources, some portion of the air emissions contributes to mercury levels in urban runoff, which in turn may contribute significant amounts of mercury to the aquatic environment and POTWs, 3.5-350 kkg and 0.8-80 kkg, respectively.

B. PRODUCTION

1. Introduction

Mercury can be detected in the earth in nearly all crustal deposits in concentrations ranging from parts per billion to parts per million. For an ore deposit to be economically desirable, the ore must contain at least 1.8 kg of mercury per metric ton (MT) of rock. Cinnabar (mercuric sulfide, HgS) is the principal ore of mercury. Mercury deposits in the United States are primarily located in Nevada and California, and seven mines were in operation in 1976: New Almaden, Oat Hill, Manhattan-One-Shot, Aetna and Knoxville in California and McDermitt and Carlin in Nevada. At the Carlin mine, mercury is recovered as a coproduct of gold refining (Van Horn 1975).

In recent years the mercury mining industry has shown great variability. In 1971 there were 71 mines in operation, which produced 616 kkg of mercury. After 1971 most of the mines discontinued their operation when the mercury price dropped and because they were unable to meet air quality standards. Mercury production in the United States fell to 75 kkg in 1974. In May 1975, a new mine with an annual capacity of 700 kkg became active in Nevada and mercury production increased to 254 kkg in 1975 and 797 kkg in 1976 (Bureau of Mines 1976). (In 1979, only the McDermitt mine was producing.) As of 1980, there are only two producing mercury mines, both in Nevada (Bureau of Mines 1979, 1980).

Secondary production of mercury provided 116 kkg of mercury in 1976. Over 60% (1515 kkg) of mercury consumed in the United States in 1976 was imported.

2. Process Description

Mercury ore is obtained from open pit surface mines and underground mines. The ore is crushed, sized, and then fed from the storage bin into a rotary kiln or a retort, where it is heated. The mercuric sulfide is decomposed at about 96% efficiency. The mercury vapors are passed through a condenser, where they are cooled below the dewpoint to form liquid mercury. The mercury is then bottled in 76-lb flasks (Van Horn 1975).

The McDermitt mine in Nevada employs a new technology. The ore, which has a very high mercury content (about 4.6 kg per metric ton of ore) is crushed and sized and then concentrated by flotation techniques. The concentrate from the flotation operation is fed through a six-hearth furnace, where the mercury in the concentrate is vaporized. The mercury vapors are cooled, condensed, sent through a cleaning process, and stored in 1000-kg shipping containers (Van Horn 1975).

The new technology used in the McDermitt mine is claimed to minimize the mercury discharges to the environment. All of the process water is recycled. Water used in the condensers is non-contact water and is discharged or recycled. The tailings from the flotation process are discharged to a pond for permanent storage. Leaching from the tailings does not appear to present a problem. Exhaust gases containing sulfur dioxide and traces of mercury vapor are discharged through a low stack, and the mercury content in these vapors is not allowed to exceed the 2.3-kg per day EPA compliance level (Van Horn 1975).

3. Environmental Releases from Mining and Production Processes

Aquatic discharges from mercury mining are estimated to be zero (Van Horn 1975, Calspan 1979) because, though there are no aqueous discharges associated with mercury mining processes, there is also very little rainfall in the Western states where mercury is mined; therefore, leaching and runoff are considered insignificant. Air emissions from secondary production facilities are estimated to amount to 7.8 kkg, and solid waste is estimated to be 0.4 kkg (Van Horn 1975). None of the secondary production facilities has an effluent discharge (U.S. EPA 1979a); therefore, the discharge to water is estimated to be zero.

In addition, there are numerous inactive or abandoned mercury mines in the United States. Release of mercury from these sites is improbable since little mercury is expected to be found in the tailings of a mercury mine (Martin and Mills 1976).

C. USES

1. Introduction

Mercury use in the United States is given in Table 2 for the period 1965-1978. As can be readily seen, the pattern of mercury use in the U.S. has been significantly altered over this period. The probable reasons for such changes are the wide publicity given to the toxic and pervasive nature of mercury and its compounds, and the availability of feasible alternatives and substitutions. However, in use areas in which there are no feasible alternatives, total mercury use has been relatively constant or has increased.

2. Electrical Apparatus

a. Introduction

The product category of electrical apparatus, which consists of batteries, lamps, switches, and rectifiers was the largest single consumer of mercury in 1976. Consumption in this category rose to 948 kkg, an increase of 62% over 1975 consumption. This increase was primarily due to the surge in use of smoke detector devices, many of which use mercury cells. Compared with total mercury consumption, however, emissions from this category during manufacturing are small.

TABLE 2. CONSUMPTION OF MERCURY IN THE UNITED STATES, 1965-1978

End Use	Consumption (kkg)[1]													
	1965	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976	1977	1978
Agriculture	107.4	81.8	128.6	118.2	92.7	62.4	50.9	63.3	63.1	33.8	20.7	20.9	20.1	W[2]
Amalgamation	9.2	8.5	7.5	9.2	6.7	7.5								-
Catalyst	31.8	66.6	85.8	66.0	102.0	77.1	34.9	27.6	23.2	44.7	23.7	43.6	53.2	W[2]
Dental preparations	110.2	73.6	82.2	106.1	99.3	78.8	81.4	102.8	97.3	104.2	61.1	68.6	42.4	17.6
Electrical apparatus	651.0	607.9	559.2	676.6	637.3	549.8	582.0	636.1	620.4	678.2	559.6	947.7	1005.7	598.1
Electrolytic preparation of chlorine and caustic soda	301.7	397.8	493.1	601.6	714.2	517.4	418.9	397.0	450.5	582.4	524.7	553.3	370.3	384.8
General laboratory use	80.4	76.4	66.9	68.6	66.7	62.2	62.0	20.4	22.7	16.4	9.6	20.5	14.0	14.5
Industrial and control instruments	356.0	251.4	257.1	275.0	229.4	166.5	167.9	225.4	246.6	213.8	141.4	174.6	180.0	120.3
Paint: antifouling	8.8	4.8	5.2	13.5	8.4	6.8	14.3	1.1	1.1					
Paint: mildew-proofing	283.0	302.9	242.2	350.7	327.0	349.9	282.3	282.3	261.0	234.6	238.7	207.4	288.3	308.9
Paper and pulp manufacture	21.3	21.1	15.4	14.4	19.2	7.8	0.1	0.03						
Unknown	-	-	-	-	-	-	-	-	-	-	-	33.4	-	-
Pharmaceuticals	14.4	8.0	9.8	14.6	24.5	23.8	23.5	19.9	20.9	20.6	14.9	2.1	W[2]	W[2]
Other	560.1	563.8	443.1	285.2	314.8	209.7	83.1	147.5	69.2	121.1	59.8	100.7	89.2	216.1
TOTAL	2,535.4	2,464.7	2,396.1	2,599.6	2,666.8	2,119.8	1,801.1	1,823.6	1,871.0	2,050.1	1,761.4	2,235.8	2,063	1,660

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[1] Original sources of these data reported mercury volumes in units of 76-lb flasks; these units have been converted to kkg by use of the factor of 0.0342 kkg/flask.

[2] W - Withheld to avoid disclosing confidential information.

Sources: Van Horn (1975); Cammarota (1975); Personal communication, H.J. Drake, Bureau of Mines (for 1976-1978).

Only about 10 kkg of the mercury are estimated to be lost to the environment during manufacturing processes. The balance of the mercury consumed by this category (938 kkg) is in the manufactured products.

These manufactured products vary both in their useful lifetimes, and in the durability of their construction. No detailed studies have been conducted in order to determine the rate of mercury release to the environment, but it is possible to make some reasonable estimates to bound the problem. By far the largest portion (80%) of mercury used in this category goes into batteries (see Note 16 to Table 1) with the "lamps", and the "tubes and switches" subcategories each accounting for 10%. As explained in the following paragraphs, rough assumptions about the use and disposal of these products suggests the following approximate releases (Arthur D. Little, Inc., estimate):

	Release (kkg)			
	Air	Water	Land	Total
Batteries	112.6		637.8	750.4
Lamps	30.6	15.6	47.5	93.7
Switches, etc			93.7	93.7
Total Released	143.2	15.6	779.1	937.8

About 94 kkg of mercury is consumed in the manufacture of mercury lamps of all types (10% of the total in this category), and for this analysis no recycling is assumed to occur. Since no data were found with which to estimate releases, a number of assumptions were made for the purposes of providing perspective on the sources. It was first assumed that one-half of the mercury used in lamps goes to mercury vapor street lamps. It was assumed that these are replaced only when they are broken, and have released one-third of their contents to the atmosphere, and the remaining two-thirds either to the soil or pavement, contributing to surface runoff. It was further assumed that the remaining one-half of the mercury used in lamps goes to production of lamps for indoor use, which are not broken before replacement. After they are replaced, these lamps may be broken in trash barrels, in transit to waste disposal centers (landfills or incinerators) or at the landfills. In this process it was assumed that 20% of the mercury is released directly to the atmosphere and that of the remaining 80%, 15% is incinerated (all of which goes to the atmosphere), and 85% goes to landfills. These assumptions result in the following estimated releases (Arthur D. Little, Inc., estimate):

	Release (kkg)		
	Air	Water	Land
Street lamps	15.6	15.6	15.6
Indoor lamps breakage	9.4		31.9
incinerated	5.6		
Total Released	30.6	15.6	47.5

b. Batteries

Mercury is used in the manufacture of zinc-carbon dry cells, carbon-zinc air cells, alkaline-manganese dioxide dry cells, mercury cells, (Ruben, Weston, and mercury-cadmium cells), and zinc-silver oxide cells. The zinc-carbon dry cell dominates the primary battery market. However, new superior electrode materials will decrease demand for the standard zinc-carbon batteries, and production of alkaline-manganese dioxide batteries and mercury cells will probably increase (Versar 1975).

In the standard zinc-carbon cell, mercury is used in the form of an amalgam with the zinc components in order to reduce corrosion of the zinc and the subsequent electrical shorting that the corrosion products could cause. In the carbon-zinc air cells, mercury is also amalgamated with the zinc component, which is used to make the electrodes, and, in the alkaline-manganese dioxide dry cells, mercury is used in combination with zinc to make corrosion-resistant anodes (Versar 1975).

Mercury cells are classified according to three types: the Ruben cell, the Weston cell, and the cadmium-mercury cell. In the Ruben cell, the cathode consists of mercuric oxide and graphite and the anode is zinc-mercury amalgam. In the Weston cell, the anode is made of cadmium amalgam and the cathode is made of mercury metal. The mercury-cadmium cell is very similar in construction to the Ruben cell, but with certain proprietary changes in the electrode composition; the anode consists of mercuric oxide and the cathode is of cadmium that is amalgamated with up to 20% mercury by weight (Versar 1975).

The zinc-silver oxide dry cell is similar to the Ruben cell, except that only the anode contains mercury and it is in the form of zinc amalgam (Versar 1975).

Rejected batteries are the major waste products generated by battery manufacturing operations. Some of the mercury is reclaimed from these batteries and the remaining is landfilled. It is estimated that only a minor amount of mercury is lost to air (0.2 kkg) and water (0.1 kkg) during battery manufacturing. Disposal of used batteries has been estimated to result in releases of about 113 kkg to air and 638 kkg to land.

c. Electric Lamps

Mercury is used in the manufacture of fluorescent, mercury vapor, metal halide, and high-pressure sodium lamps. These lamps are primarily used in street lights, high-ceiling rooms, motion picture projectors, photography, dental examinations, photochemistry, heat lamps, and water purification. GTE-Sylvania, Westinghouse Electric, General Electric, and North American Philips are the largest manufacturers of these lamps (Van Horn 1975).

Mercury lamps are produced by injection of the liquid mercury, vapor, a starting gas, and other materials into a sealed quartz tube. Mercury losses during manufacturing are relatively small. Substantial mercury losses to the environment occur when consumers discard the spent tubes. In all 0.2 kkg is estimated to be released to the atmosphere and 3.6 kkg to landfills.

d. Switches, Rectifiers, etc.

No information is available concerning the amount of mercury released during manufacturing and use of this product. However, its characteristics suggest a relatively long lifetime, no recycling, and ultimate disposal of all 93.8 kkg of the mercury consumed each year for this use in a landfill.

3. Electrolytic Preparation of Chlorine and Caustic Soda

The second largest user of mercury is the mercury-cell segment of the chlor-alkali industry. In this process, mercury serves as a flowing cathode for the electrolytic decomposition of salt brine into chlorine, sodium hydroxide, and hydrogen. Theoretically, the mercury can be used repeatedly without any losses. However, mercury losses do occur and mercury is replaced at a rate of about 0.7-1.0 kg of mercury per metric ton of chlorine.

A typical mercury-cell plant produces 270 kkg (300 tons) per day. The smallest plant produces 94 kkg/day (104 tons/day) of chlorine; the largest 600 kkg/day (725 tons/day). Figure 3 shows the locations of mercury-cell chlor-alkali plants in the U.S.

Airborne mercury emissions from mercury-cell chlor-alkali plants consist of mercury vapor in cell room air and mercury loss along with byproduct hydrogen gases, which are scrubbed and filtered to remove most of the mercury. In the mercury-cell process, the mercury flows through the cells as a flowing cathode, and in order to maintain the flow, mercury must be added at one end of the cell and removed at the other end by means of inlet and outlet "end boxes." The end boxes are usually kept under a slight vacuum, and the chlorine-containing gases that are captured and scrubbed from the end boxes usually contain some elemental mercury that is not completely recovered (Versar 1976a).

Present National Emission Standards for Hazardous Air Pollutants (NESHAP) require that no more than 2,300 g per day of mercury vapor be lost to the atmosphere from each mercury-cell plant regardless of size. In practice, all plants cool the hydrogen to a temperature of 13°C (55°F) or less to condense and recycle most of the mercury in this stream. Most mercury plants further treat the hydrogen with activated carbon or molecular sieves that amount to but a few grams a day (Versar 1976a).

Three types of solid wastes are generated from the mercury-cell chlor-alkali plants (Versar 1976a):

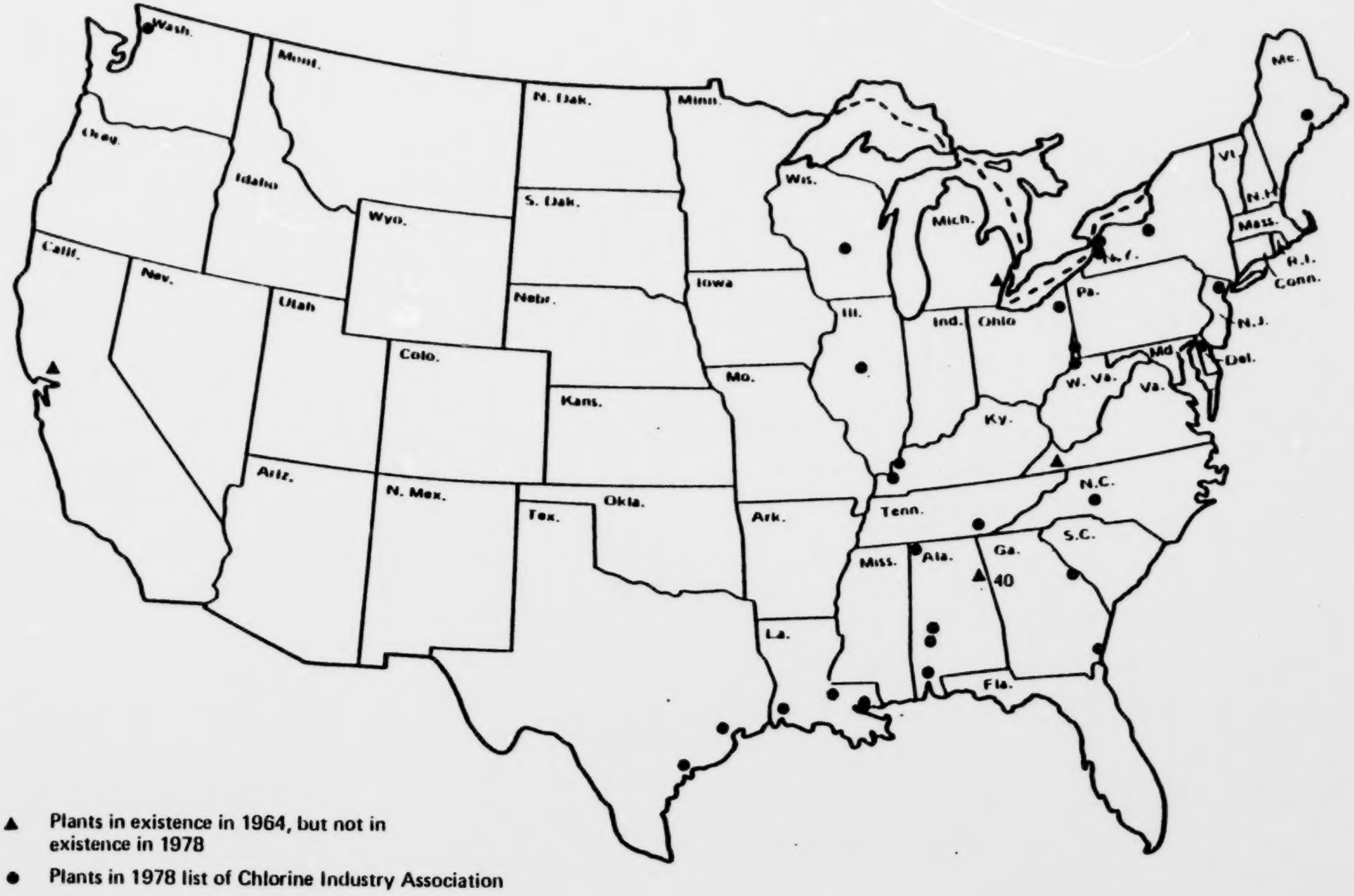

FIGURE 3 LOCATION OF MERCURY CELL CHLOR–ALKALI PLANTS IN THE U.S.

(1) the brine purification sludge that primarily consists of calcium carbonate and magnesium hydroxide, with minor amounts of sodium chloride and mercury. The mercury is present either in elemental form or as a complex $HgCl_4^=$;

(2) mercury cell "butter," which is primarily an amalgam; and

(3) mercury spills and cleanouts, which are flushed into sumps below the cell room floor to become waterborne wastes.

No waterborne wastes are generated from the mercury-cell operation itself. However, wastewaters from mercury-cell plants are generated from collected mercury spills, cell end-box purges, cell washings, and brine leaks and spills. Due to the very low levels of mercury allowed in plant wastewater, cell plants have wastewater treatment for mercury removal. Available data from 1976 show that the mercury discharge at every plant having effluent outfalls is within the applicable NPDES permit range of 0.013 to 0.35 kg/day for BPT (best practicable treatment) and the Effluent Limitation Guideline which is generally given in kg/kkg of product (Versar, Inc. 1976a).

Over 530 kkg of mercury is discharged to land annually by chlor-alkali plants. The number of plants has remained relatively stable over the past 15 years, and the major growth in the chlorine industry has occurred using technology that does not require mercury cells. However, there are apparently nearly 30 sites in the country that have accumulated large amounts of solid waste material from mercury-cell plants, some of which are no longer active. No estimate can be made of the mercury that is contained in these disposal sites. In addition, these areas represent a potential source to the aquatic environment, although the extent of releases is unknown.

4. Industrial Instruments

Mercury is used in the manufacture of switchgear and switchboard apparatus and mechanical measuring and control instruments because of its high thermal conductivity and well-known thermal expansion properties. Some of the major types are thermometers, thermostats, thermoregulators, flowmeters, pressure-sensing devices and barometers, gages, valves, pump seals, switches and relays, navigational devices, and medical devices.

In the manufacturing of industrial instruments, mercury is lost to the environment from spillage, from volatilization, and in cleaning of old instruments that require servicing. The majority of the mercury losses to the environment, however, occur from disposal of damaged consumer products containing mercury (Van Horn 1975). as is shown in Table 1.

5. Mercury Compounds

a. Introduction

Mercury compounds are used in the production of fungicides and bactericides, catalysts, paints, and pharmaceuticals and in general laboratory use.

The manufacturing process for mercury compounds involves conversion of the metal into one of several inorganic mercurials that are appropriate intermediates to the production of other inorganic or organic compounds. There are about 16 manufacturers of mercury compounds in the United States. Often the same facility produces both inorganic feedstock and organic mercurials (Van Horn 1975).

Mercury compounds are low-volume chemicals. The most common mercurials are mercuric chloride, red and yellow mercuric oxide, mercurous chloride, and aminomercuric chloride.

b. Catalysts

Manufacture of catalysts consumes 43.6 kkg of mercury each year. This is a small but important use of mercury compounds. Mercury catalysts are used in the synthesis of vinyl chloride and vat dyes.

Vinyl chloride monomer produced from acetylene and hydrogen chloride uses a mercuric chloride catalyst. Most of the mercury losses occur when carbon pellets impregnated with mercuric chloride are discarded (Van Horn 1975).

Anthroquinone vat dyes are sulfonated in the presence of mercuric sulfate to yield two dyestuff intermediates (1,5 and 1,8-disulfonic acid). These vat dyes are easily reduced to a soluble, colorless form in which the fibers are readily impregnated; the dyes are then oxidized to produce the insoluble color in the fibers (Van Horn 1975).

Environmental releases from catalysts manufacturing each year are estimated to amount to 0.1 kkg to air and 0.2 kkg to water. Losses of mercury to the products of catalyzed reactions are considered to be insignificant. The bulk of the releases from this category is from the disposal of spent catalysts, all of which are assumed to be disposed of in land, since recovery of mercury from mercurial catalysts is not economically feasible.

c. Paint Manufacturing

The paint industry consumed about 12% of the mercury produced in 1976. A small amount of this mercury was used for mildew-proofing substances; the remainder was used in paint additives. Phenylmercuric compounds, primarily phenylmercuric acetate, are used as in can preservatives in water-based paints and coatings at levels of 50-100 mg/l.

Exterior water-based paints may contain phenylmercuric acetate as a mildewcide at levels of 250-1500 mg/l to prevent fungus growth on the applied paint film (U.S. EPA 1976a). Although the mercury levels in paints are low, large quantities of paints are used, so that the amount of mercury involved is quite significant.

Paint manufacturing plants are estimated to discharge 0.4 kkg to POTWs each year. Airborne emissions and solid wastes are considered to be negligible. However, mercury emissions from paint applications are expected to equal the mercury content. Air emissions are expected to be 65% after application. The remaining 35% is expected to be distributed between incineration (air), landfill, land fallout (from paint peeling), and runoff.

d. Fungicides and Bactericides

In 1970, USDA banned the use of all alkylmercury pesticides. As a result, phenylmercuric acetate (PMA) became the major organomercurial used in agriculture. In August 1976, the U.S. EPA passed a regulation that allows for the temporary use of mercury biocides to treat summer golf turf diseases and certain farm seeds (barley, wheat) until August 31, 1978, or when the equivalent of 2 years' production of the latter biocides has been attained, but as of that date these uses were cancelled. Use of mercury-based biocides on winter golf turf diseases is allowed by the U.S. EPA under strictly controlled conditions (U.S. EPA 1966).

Mercury lost to the environment during the manufacturing process is expected to be negligible. The bulk of the mercury in pesticides reaches the land upon application. Losses to water occur from the leaching of mercury into groundwater supplies and from rain runoffs.

e. Pharmaceuticals

The use of mercury in pharmaceuticals has decreased greatly in recent years. Organic mercury compounds are used in diuretics and antiseptics. Inorganic salts are used in solutions to sterilize instruments. Ammoniated mercury, mercury oxides, and metallic mercury are used in skin preparations. Phenylmercury compounds are used as preservatives in cosmetics and soaps (Van Horn 1975).

Due to the decline in mercury use, environmental discharges from the manufacturing operations are judged to be negligible. The bulk of the mercury released to the environment from pharmaceutical use is likely discharged primarily to POTWs, since mercurials are discarded from the body along with other waterborne body wastes.

f. General Laboratory Use

Metallic mercury and mercurial compounds are used for many general laboratory purposes: as reagents and indicators, for calibration and sealing, and occasionally in vacuum pumps. In hospitals, mercury is used for diagnosis by means of radioactive markers and as a fixative for tissues. In 1976, 20.5 kkg of mercury were consumed for general laboratory uses (Van Horn 1975).

The distribution of mercury losses among particular environmental media reflects primarily the type of the use of the chemical rather than the type of laboratory (Van Horn 1975). Mercury is lost to the atmosphere through volatilization; mercury is lost to POTWs as a result of discarding the chemicals down the drain when the experiment has been completed; some mercury is also lost from spillage and from washing glassware that contained mercury compounds. Most laboratories, however, have instituted techniques for recycling mercury wastes. It is expected that between 55% and 60% of the mercury that is consumed is recycled.

g. Dental Preparations

Mercury is used as an amalgam in dental work. Fillings are prepared by combining a silver-tin alloy in powdered form with metallic mercury. A metallic putty is formed composed of silver-mercury, tin-mercury compounds, and some residual silver-tin alloy. This putty is then placed in the drilled tooth cavity. The putty is condensed and polished and the excess amalgam is removed by filing during the condensing process. This excess amalgam is normally released to the wastewater system unless a trap to catch the amalgam is installed on the drain (Van Horn 1975).

"Premixes" are now available in which the mercury and silver-tin alloy are in measured quantities to prevent the losses of mercury during the preparation of the filling (Van Horn 1975). It is estimated that 22% of the mercury consumed is lost to water and 2% is lost to air during application of dental preparations. The remainder of the mercury is in the dental work of the patients. However, it can be assumed that the remainder will eventually be lost to the environment as well.

D. NATURAL AND INADVERTENT SOURCES

Approximately 40% of the total identified mercury released to the environment is from natural sources and 11% is from inadvertent sources, such as mining operations and fossil fuel combustion.

1. Natural Sources

Mercury is naturally present in the environment as a result of the outgassing of the earth's crust and as runoff from natural erosion processes. Natural processes add mercury to the biosphere at a constant rate. In the United States, the outgassing process releases over 1000 kkg of mercury (approximately 60% of the identified total mercury released to this medium) to the atmosphere. Runoff contributes almost 200 kkg of mercury to the water (approximately 63% of the known mercury released to the water). These discharges of mercury are quite large, but they are widely distributed in the U.S., although mercury deposits and mineralized areas are concentrated in the western U.S., resulting in higher rates of degassing from this region (Van Horn, 1975).

2. Fossil Fuel Combustion

Fossil fuel combustion contributes about 5% (~190 kkg) to the identified mercury released to the environment. The bulk of this emission (180 kkg) is airborne. The concentration of mercury in coal and other fuels is very low (0.066 mg/kg to 0.2 mg/kg) (Van Horn 1975). The releases of mercury to the air via combustion of fossil fuels are substantial only because of the enormous quantities of fuels burned. The airborne emissions of mercury are primarily in the metallic form. Land-destined wastes of mercury from these operations result from the disposal of the bottom ashes.

3. Mining and Smelting Operations

Since mercury is present as an impurity in the ores of other metals and minerals, it enters the environment as a byproduct of mining and smelting operations.

Verification data from the Effluent Guidelines Division indicate that coal mines discharge 3.2 kkg of mercury each year to the waters (U.S. EPA 1979a). Other mining and smelting operations contribute 0.9 kkg to water (Van Horn 1975). Airborne emissions and solid waste discharges from this category are 50.8 kkg and 5.1 kkg, respectively (Van Horn 1975).

The gold milling process originally used mercury amalgam plates to recover gold particles, although this process has been replaced by cyanide leaching. While no releases of mercury can be attributed to this source, the past use of mercury may represent a source of exposure through highly contaminated sediments in the vicinity of gold mining areas (Martin and Mills 1976).

4. Mercury as an Impurity

Approximately 191 kkg of mercury enter the environment as a result of its occurrence as an impurity in fertilizers. Some of this mercury may also enter the aquatic environment as a result of runoff.

The Effluent Guidelines Division's screening and verification data (1979c) reveal mercury in the effluents of the Timber Products Processing Industry, Petroleum Refining, and Auto and Other Laundries. Mercury is not expected to be used by these industries; therefore, it must be present in the effluent as an impurity. These industries have been estimated to discharge 0.7 kkg of mercury to waters and 0.7 kkg to POTWs.

5. Publicly Owned Treatment Works

The amount of mercury found in sewage effluents and sludges varies greatly. It has been estimated that 42.2 kkg are discharged to the aquatic environment (see Note 25, Table 1). Sources to POTWs include domestic and industrial waste, and urban runoff.

An estimated 3.6 million kkg of sewage sludge solids containing between 0.36 and 203.2 kkg of mercury are collected annually in the United States (SRI 1979). About 25% of this sludge is applied to land as a result of the sale of sludge for use as commercial fertilizers or soil conditioners. The remainder is disposed of by incineration (35%), in landfills (25%), and in the ocean (15%) (SRI 1979).

6. Urban Runoff

Mercury has been found in urban runoff at levels of about 0.2-85 ug/l (Murphy and Carleo 1978). The mean value for a residential area of 720 acres in Rochester, N.Y. was found to be 18.1 ug/l with the median value for the same set of 10 data points in the range of 4-5 ug/l. A second study involving less intensive sampling of stormwater and combined sewer runoff in 11 cities across the U.S. (including Rochester, N.Y.) revealed concentrations ranging from less than 0.2 ug/l to 0.6 ug/l (Turkeltaub and Weisman 1981). The mean and median values for this data set were both equal to 0.3 ug/l. (The mercury concentration reported for Rochester in this study was 0.25 ug/l.) Lacking further information. a range of 0.2-20 ug/l in urban runoff was used to show the possible magnitude of the source. Thus, at volumes of runoff of 17.3 x 10^{12} l/yr and 3.6 x 10^{12} l/yr going to surface waters and POTWs respectively (U.S. EPA 1977a), 3.5-350 kkg goes to surface waters, and 0.8-80 kkg to POTWs each year.

The most likely sources of mercury in urban runoff are air emissions (subsequently deposited) from smelters. incinerators. fossil fuel combustion, and various other industrial activities, as well as mercury volatilized from paint. Since these releases have already been included in the materials balance shown in Table 1, the urban runoff figures have not been included in Table 1.

E. CONCLUSIONS

The published literature provides very little information regarding the amounts and the fate of mercury wastes resulting from manufacturing operations and from use of mercury-containing products. In general, it appears that considerably more effort has been devoted to assessing the amounts and fates of mercury wastes resulting from manufacturing operations than to those from the application of mercury-containing products, in spite of the fact that manufacturing wastes may represent only a minute quantity compared with potential wastes from manufactured industrial and consumer products, as indicated by the following:

Industry	Mercury Input (kkg)	Segment of Industry	Amount Hg to Environment (kkg)
Electrical Equipment	948	Manufacturing	9.9
		Products	938.1
Paint	270	Manufacturing	3.2
		Products	266.8

For both industries, the fate of relatively small amounts of waste mercury from manufacturing operations is known with what is judged as fair reliability. The disposition of the mercury in these products has been estimated as indicated in Table 1 and as explained in the notes to Table 1. In view of the fact that the total consumption of mercury in the U.S. in 1976 was about 2400 kkg, the inability to allocate reliably the fate of approximately 1200 kkg in two classes of products mentioned above should be noted.

A more complete materials balance for mercury in the United States could be prepared if the following information were available:

(1) The product mix of short-lived and long-lived consumer and industrial products containing mercury.

(2) The failure or replacement rate for products that will last beyond the year of their manufacture.

(3) The fate of both short-lived and long-lived products containing mercury.

In view of the data available, and considering the assumptions made, the analysis presented here still provides insight into the identification of sources of mercury to the environment. Of the identified releases from natural and anthropogenic sources, about 48% went to land, 43% to air, 8% to water, and 1% to POTWs. Sources to air are largely natural (about 60%) of air releases, while paint volatilization and fossil fuel combustion contribute 12% and 10% to air, respectively. Sources to water are also largely natural (about 63%) as a result of soil erosion. POTWs contribute about 14% of mercury loadings directly to water, while dental preparations, paint applications and electrical apparatus each contribute 5-8% to water. Sources to land are largely attributed to disposal of electrical apparatus (42%) and wastes from chlor-alkali plants (27%). Mercury in fertilizer, disposal of POTW sludges, and disposal of industrial and control instruments comprises 5-10% of the mercury reaching the land compartment. In addition, it was calculated that 3.5-350 kkg mercury may be contained in urban runoff. The higher value suggests that a significant portion of mercury initially reaching the air or land compartment eventually reaches the aquatic environment directly from storm sewers or through POTWs.

As was discussed previously, numerous uncertainties exist in this analysis. The largest errors are probably in the estimation of mercury as a natural source, of mercury released from product use and disposal, and of mercury contained in urban runoff.

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CHAPTER IV.

FATE AND DISTRIBUTION OF MERCURY IN THE ENVIRONMENT

A. MONITORING DATA

1. Introduction

The following collection of data represents levels of mercury observed in the U.S. environment from 1970 through 1979, gathered from published reports and computerized monitoring data bases. Levels of mercury have been measured in water, sediment, rocks and soils, the atmosphere, and terrestrial and aquatic biota, and the available data concerning concentrations in these media are presented in the following sections. Natural sources appear to account for a significant proportion of the mercury in the environment. Mercury levels in all media tend to be higher in urban areas than in rural areas. Studies that enable a comparison of environmental mercury levels over time indicate a perceptible reduction in mean levels and a significant leveling off of mercury levels in locations with high levels, due to reductions in releases to the environment in many industrial sectors. A tremendous amount of monitoring data is available for mercury. However, only a few examples are included here. For more detailed discussions, the reader is referred to Battelle (1977) and WHO (1976).

2. Water

Numerous analyses of mercury levels in water have been conducted. Table 3 summarizes the results of a few studies.

Studies of uncontaminated ocean waters (Williams and Weiss 1973, Hosohara 1961) found mercury concentrations ranging from 0.029 ug/l to 0.27 ug/l. A reported mean mercury level in oceans of 0.1 ug/l (Goldberg 1972) is consistent with this data.

Mercury concentrations in freshwater are similar to those found in oceans. Various studies support the observation that mercury levels in uncontaminated streams, rivers and lakes are usually between 0.01 ug/l and 0.1 ug/l[1] (Jonasson and Boyle 1971). Garcia and Kidd (1979) found an average of 0.027 ug/l mercury in New Mexico's

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[1] Values above 0.1 ug/l are probably caused by natural or anthropogenic contamination (Jonasson and Boyle 1971).

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TABLE 3. CONCENTRATIONS OF MERCURY DETECTED IN WATER

Location	Concentration (ug/1)	Source	Comments
Ocean	0.04-0.27 (range)	Williams and Weiss (1973) Hosohara (1961)	Uncontaminated areas
Ocean	0.1 (mean)	NSF (1972)	Uncontaminated areas
Freshwater	0.01-0.1 (range)	Jonasson and Boyle (1971)	Uncontaminated areas
New Mexico (reservoir)	0.027 (mean)	Garcia and Kidd (1979)	Uncontaminated area; 116 samples
England (lake)	0.12-0.029 (range of mean values)	Gardner (1978)	Uncontaminated area; 116 samples
Great Lakes	.16 (mean) 0.0-0.4 (range)	Chau and Saitoh (1973)	
Northern Mississippi Streams and Lakes	0.28 (mean)	Rihan *et al.* (1978)	Agricultural area with mercurial fungicides; 10 samples
Minimata Bay, Japan	1.6-3.6 (range)	Hosohara *et al.* (1961)	Contaminated area
Rochester, NY urgan runoff	18.1 (mean)	Murphy and Carleo (1978)	Urban area

second largest reservoir. In England, Gardner (1978) reported mean mercury concentrations in an uncontaminated lake ranging from 0.012 ug/l to 0.029 ug/l.

Slightly higher levels of mercury are found in freshwaters located near possible sources of contamination. In a two-year study of the Great Lakes, Chau and Saitoh (1973) reported a mean mercury concentration of .16 ug/l, with values ranging from undetected to 0.4 ug/l. Rihan et al. (1978) studied streams and lakes in northern Mississippi, an agricultural area where mercurial fungicides are used. The average mercury level in these waters was 0.28 ug/l.

Water in highly industrialized and/or contaminated areas may have greatly elevated mercury concentrations. In Minimata Bay, a contaminated area in Japan, mercury concentrations were reported to range from 1.6 ug/l - 3.6 ug/l (Hosohara et al. 1961). Murphy and Carleo (1978) report average mercury levels of 18.1 ug/l in urban runoff in Rochester, N.Y.

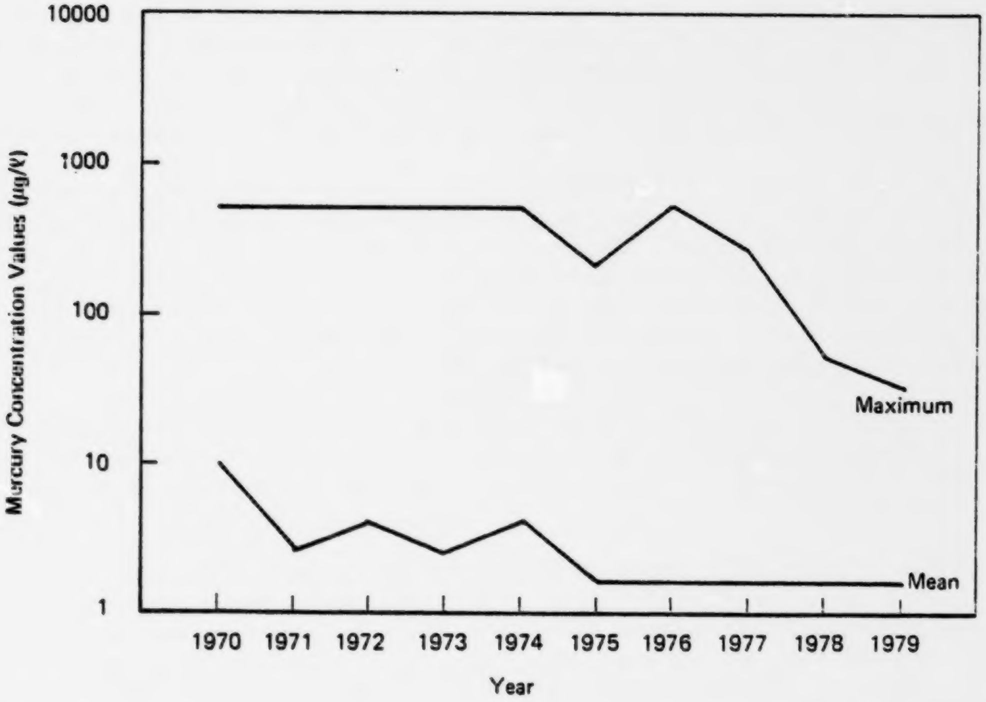
The STORET water quality data system provides sampling information, which indicates the distribution of reported ambient concentrations of mercury in surface water. These data were retrieved for the time period 1970 to 1979 and aggregated on a national level and for major river basins. Trends in ambient concentrations were investigated over the past 10 years for both the continental United States and the geographical regions delineated by major river basins.

The 10-year trend of maximum and mean concentration values for the United States is presented in Figure 4. During this period, over 100,000 observations of total mercury were made at roughly 13,000 water quality stations.

From 1970 to 1974, mean mercury concentration values over the U.S. exceeded 1.0 ug/l annually. Since 1975, the annual mean concentration values have fallen to between 0.5 ug/l and 0.7 ug/l. Likewise, sampling records prior to 1975 show maximum values over 500 ug/l in several areas in the country. These maximum levels decreased such that these localized maximum concentration values typically are below 200 ug/l nationwide. These reductions may represent improved analytical techniques, but more likely reflect actual reductions in mercury levels.

In order to examine these trends in greater detail, data were examined for a total of 23 major river basins. Figure 5 illustrates a significant decrease in mean concentration values for major river basins from 1972 to 1979.

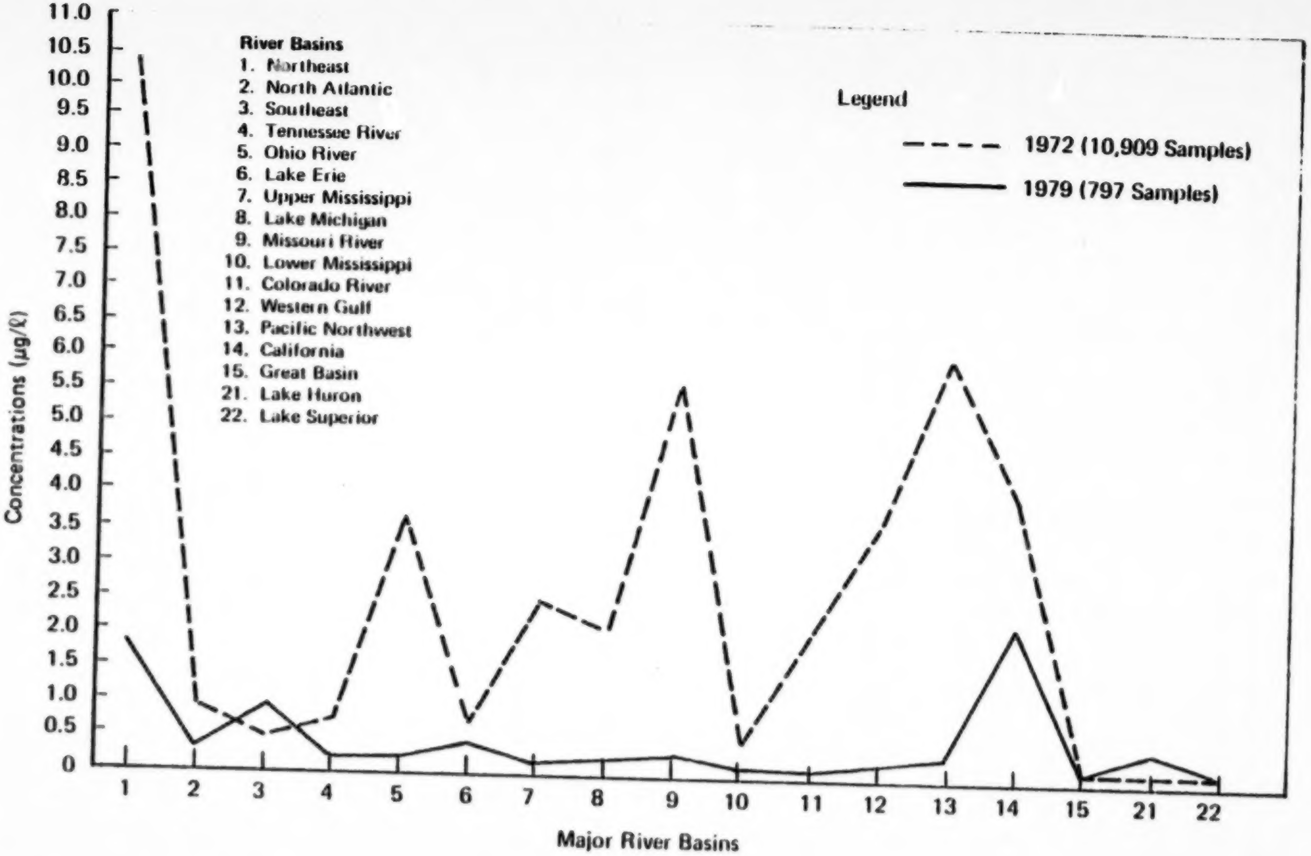
Measurements of mercury in water are usually of total mercury due to the limitations of the analytical techniques available for organic mercury. The most sensitive organic test has a detection limit of 2×10^{-11}g Hg/g water (ORPG, 1979) which exceeds typical concentrations of the compound in ambient waters. However, organic mercury levels in water may be estimated by examining other water parameters. Using a partition



Number of Observations: 107,016
Number of Stations: 13,443

Source: STORET

FIGURE 4 MAXIMUM AND MEAN AMBIENT CONCENTRATIONS OF MERCURY IN SURFACE WATERS OF THE UNITED STATES, 1970-1979 – A TEN-YEAR TREND



Source: STORET

FIGURE 5 MEAN LEVELS OF MERCURY IN MAJOR RIVER BASINS IN 1972 AND 1979

coefficient of sediment to water of 170[2] (Akagi et al., in press), organic mercury levels in the Ottawa River were estimated to be approximately 0.0018 ug/l, which is 24% of the total mercury (ORPG, 1979). Preliminary laboratory studies revealed organic fractions of 10-20%, which are consistent with the preceding estimate (ORPG 1979). How typical these fractions are of aquatic systems in general is unknown due to a lack of sampling data for verification.

3. Sediment

From the STORET data retrieved, mercury concentrations in sediment were not continually declining from 1970 to 1978 as were concentrations in water. However the mean concentration dropped from 8 mg/kg in 1970 to 3 mg/kg in 1972 and remained below 3 mg/kg until 1978. Figure 6 exhibits the maximum and mean concentrations of mercury in sediment from 1970 to 1978 for the United States.

Table 4 briefly summarizes mercury concentrations reported in sediment. Mercury concentrations in sediments near sources of contamination are higher than those in unpolluted areas. Price and Knight (1978) found a mean mercury concentration of 0.0481 mg/kg in sediment from a relatively uncontaminated lake and reservoir in Mississippi. In contrast, Jackson (1979) found a mean mercury level of 1.27 mg/kg in the sediment of a lake downstream from an area of high industrial activity, including a chlor-alkali plant and a pulp and paper mill.

Other studies of contaminated areas also indicate high mercury concentrations in sediments. Gardner et al. (1978) studied sediments of a Georgia salt marsh located near a chlor-alkali plant. This factory had discharged approximately 1 kg mercury/day of Hg from 1966 until 1972, when the discharges were discontinued. Sediments in a nearby salt marsh contained mean mercury levels of 0.56 mg/kg in samples taken at a depth of 0-5 cm., and 0.28 mg/kg in sediments 5-10 cm deep. In Palos Verdes shelf sediments in California, samples located near a major wastewater outfall were examined by Eganhouse et al. (1978). Aqua regia digestion of sediment samples showed that they contained a mean mercury concentration of 2.54 mg/kg. Roberts et al. (1975) found similar mercury levels (mean values from 2-3 mg/kg) in sediments from Boston Harbor.

The form of mercury in the sediment represents an important issue in determining exposure. The Ottawa River Project Group (ORPG 1979) estimates that usually no more than 1% of the total mercury in sediment is in the organic form. Roberts et al. (1975) found that 0.1-0.5% of the mercury in Boston Harbor sediments was in the form of methylmercury. A study of sediment in the Florida Everglades reported that similarly low percentages (0.03-0.07%) of total mercury were methylmercury (Andren and Harnss 1973). In the Palos Verdes sediments studied by Eganhouse et al. (1978) up to 2% of the total mercury was in the organic form.

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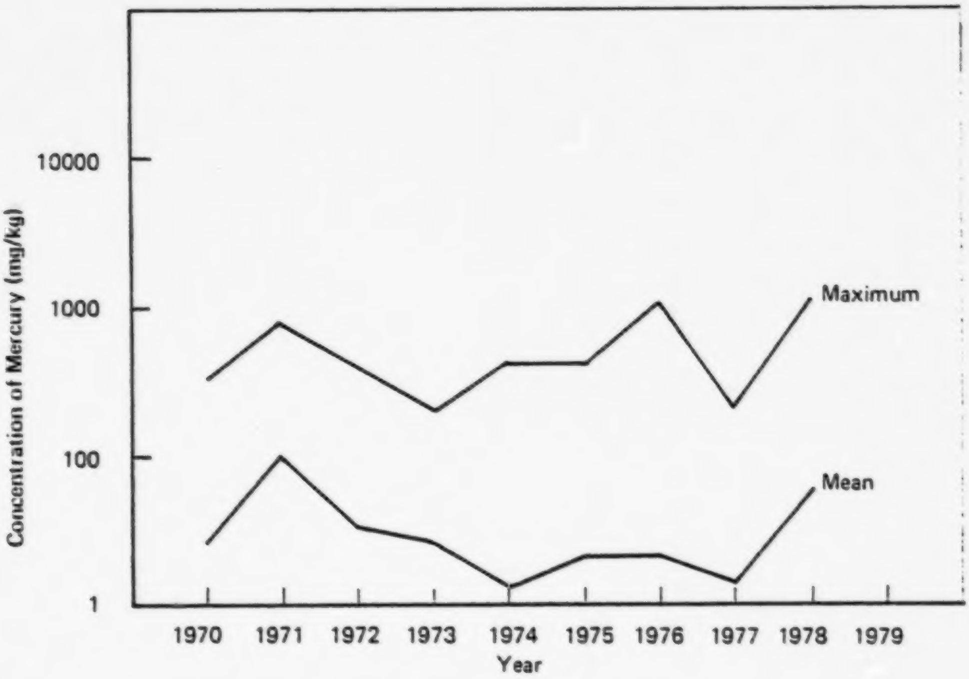
[2] For sandy sediment, using methylmercury.



Source: STORET

FIGURE 6 MERCURY CONCENTRATIONS IN SEDIMENT, UNITED STATES, 1970-1978 – A TEN-YEAR TREND

TABLE 4. CONCENTRATIONS OF MERCURY DETECTED IN SEDIMENT

Location	Concentration (mg/kg)	Source	Comments
Mississippi Lake and Reservoir	0.0481 (mean)	Price and Knight (1978)	Uncontaminated area; 19 samples
Ontario (2 lakes)	1.27 (mean)	Jackson (1979)	Contaminated areas; 18 samples
Georgia (salt marsh)	0.56 (mean at 0-5 cm)	Gardner *et al.* (1978)	Contaminated area; 10 amples at each depth
California (Palos Verdes Shelf)	2.54 (mean)	Eganhouse *et al.* (1978)	Contaminated area; samples from 14 stations
Boston Harbor	2.0-3.0 (range of mean values)	Roberts *et al.* (1975)	

Thus, it appears that methylmercury, as well as other organic forms, generally represents only a small portion of the total mercury present in the sediment.

4. Rocks and Soil

Mercury is prevalent in almost all soils and rock formations. Table 5 lists mercury concentrations found in these media. Background concentrations of mercury in these media have been reported to range 0.001 mg/kg to over 0.5 mg/kg (D'Itri 1972). Jonasson and Boyle (1971) estimate a narrower range of 0.02-0.15 mg/kg mercury for normal soils and rocks, with values as high as 250 mg/kg occurring in soils near mercury deposits.

Jonasson and Bovle (1971) studied the mercury content of various types of sedimentary, igneous, and metamorphic rocks. Their data are presented in Table 5. Since soils are derived from rocks, one would expect mercury levels in uncontaminated soils to be similar to mercury concentrations in rocks (Battelle 1977).

Data from Shacklette et al. (1971) supports this hypothesis. Soils and other regoliths were sampled throughout the United States at sites approximately 59 miles apart. The geometric mean of all mercury concentrations was 0.071 mg/kg, and 0.112 mg/kg was the arithmetic mean. Sixty-seven percent of the locations had less than 0.080 mg/kg mercury, while only 16% of the sites had values exceeding 0.175 mg/kg. The data were also segregated according to that found east and west of the 97th eridian, with geometric means of 0.096 mg/kg and 0.055 mg/kg for eastern and western soils, respectively.

Wiersma and Tai (1974) surveyed cropland and noncropland soils in 29 eastern states and found no statistical difference between the two soil types. Mean mercury levels ranged from 0.05 mg/kg to 0.10 mg/kg.

In two studies conducted as part of the National Soils Monitoring Program, mercury concentrations in 10 urban areas were compared with concentrations in the corresponding suburban areas (Gowen et al. 1973, USEPA 1974a). At 9 of the 10 sites mercury concentrations were significantly higher in the urban soils. Urban values ranged from undetected to 15.39 mg/kg while suburban levels varied between undetected and 1.12 mg/kg.

5. Air

Atmospheric mercury may exist as a vapor or associated with particulates. In addition, airborne mercury may be present in elemental, organic or inorganic forms. Therefore, monitoring data for mercury in air must be interpreted with regard to the type of mercury being measured.

Various studies indicate that atmospheric mercury is primarily a vapor (NRC 1978). However, the ratio of particulate mercury to mercury vapor may vary with the location. Airborne mercury over oceans and in

TABLE 5. CONCENTRATIONS OF MERCURY DETECTED IN ROCKS AND SOIL

Location	Concentration (mg/kg)	Source	Comments
Unspecified	0.001->0.5 (range)	D'Itri (1972)	Background concentrations in rocks and soils
Unspecified	0.02-0.15 (range)	Jonasson and Boyle (1971)	Background concentrations in rocks and soils
Unspecified	up to 250	Jonasson and Boyle (1971)	Background concentrations near mercury deposits
Unspecified	0.025-.437 (range of mean values) 0.010-3.25 (range)	Jonasson and Boyle (1971)	Sedimentary rocks
Unspecified	0.020-.450 (range of mean values) .002-1.4 (range)	Jonasson and Boyle (1971)	Igneous rocks
Unspecified	0.050-.225 (range of mean values) 0.010-1.0 (range)	Jonasson and Boyle (1971)	Metamorphic rocks
Sites throughout the United States	0.071 (geometric mean) 0.112 (arithmetic mean)	Shacklette _et_ _al._ (1971)	912 samples
29 Eastern States	0.05-0.10 (range of mean values)	Wiersma and Tai (1974)	275 cropland samples; 104 noncropland samples
10 urban areas throughout the United States	0.0-15.39 (range)	Cowen _et_ _al_. (1973), USEPA (1974a)	359 samples
10 suburban areas throughout the United States	0.0-1.12 (range)	Cowen _et_ _al_. (1973), USEPA (1974a)	477 samples

rural areas is reported to be almost completely in the vapor form, although the proportion of vaporous atmospheric mercury may vary in urban areas (NRC 1978).

Studies indicate that the major portion of airborne mercury (primarily the vapor) is in the elemental form (NRC 1978, Spittler 1976). Johnson and Braman (1974) collected air samples at eleven sites near Tampa, Florida and found different mercury species present in the vapor fraction in the following percentages: Hg(II) - 25%, methylmercury - 21%, elemental mercury - 49% and dimethyl mercury - 1% (particulate mercury comprised the remaining 4% of the sample material). Spittler's (1976) analysis of air samples in North Carolina showed even higher percentages of elemental mercury, including at least one sample that was entirely composed of elemental mercury. Soldano et al. (1974, 1975) studied airborne mercury near sewage treatment plants and found that the prevalence of certain mercury species depended upon the species and distance from the source. Levels of elemental mercury varied inversely with distance from the plant, while levels of alkyl mercury halides increased with increasing distance from the plant.

Cooper et al. (1974) report that background mercury vapor concentrations generally range from 1 ng/m^3 to 5 ng/m^3, while levels in urban areas are in the range 2-60 ng/m^3. According to the NRC (1978), typical total mercury concentrations are approximately 0.7 ng/m^3 in remote oceanic areas, 4.0 ng/m^3 in rural areas, and usually less than 10 ng/m^3 in urban areas. Table 6 summarizes these as well as other data on concentrations of mercury in air.

In the study by Johnson and Braman (1974) referred to above, total mercury levels in Tampa ranged from 1.8 ng/m^3 to 298 ng/m^3, with mean values of 4.48 ng/m^3 during the day and 8.40 ng/m^3 at night.

Spittler (1976) analyzed a variety of air samples from nine states, with most of the samples coming from the New England area. Over 90% of the samples contained mercury levels within the 2-60 ng/m^3 range reported by Cooper et al. (1974). High values appeared in the plumes of an incinerator (200 ng/m^3), a burning dump (275 ng/m^3) and a power plant (5,820 ng/m^3). Cooper et al. (1974) also reported atmospheric mercury levels in 10 cities located in the west and midwest. Values ranged from 5.0 ng/m^3 near a freeway in Nashville, TN to 29.6 ng/m^3 near an industrial area in El Paso, TX.

The mercury content of petroleum may result in transportation sources contributing significant amounts of mercury to the atmosphere. Cooper et al. (1974) found that automobile exhaust gases contained 95-160 ng/m^3 mercury. (The unburned fuel was not analyzed for mercury content.) In addition Cooper et al. (1974) report that atmospheric mercury concentrations near highways increase from 5 ng/m^3 during low-traffic periods to 10-12 ng/m^3 during periods of heavy traffic.

TABLE 6. CONCENTRATIONS OF MERCURY DETECTED IN THE ATMOSPHERE

Location	Concentration (ng/m^3)	Source	Comments
Unspecified	1-5 (range)	Cooper *et al.* (1974)	Background concentrations in rural areas
Unspecified	2-60 (range)	Cooper *et al.* (1974)	Urban areas
Unspecified	0.7 (typical value)	NRC (1978)	Remote oceanic areas
Unspecified	4.0 (typical value)	NRC (1978)	Rural areas
Unspecified	<10.0 (typical values)	NRC (1978)	Urban areas
Tampa, FL	1.8-298 (range 4.48 (daytime mean) 8.40 (nighttime mean)	Johnson and Braman (1974)	
9 States	2-60 (range for 90% of the samples)	Spittler (1976)	
10 Cities (Western and Midwestern United States)	5.0-29.6 (range)	Cooper *et al.* (1974)	
Unspecified	5 (typical value)	Cooper *et al.* (1974)	Near highway during low traffic period
Unspecified	10-12 (typical values)	Cooper *et al.* (1974)	Near highway during heavy traffic period

6. Aquatic Biota

A large amount of data concerning mercury levels in aquatic biota is available in the literature. Many of these studies have focused on the threat of human exposure from mercury in seafood. Table 7 summarizes data on aquatic biota which is discussed in this section.

Some studies of aquatic biota have focused specifically on freshwater fish. A comprehensive survey of mercury levels in freshwater fish was conducted by the National Pesticide Monitoring Program in 1969 and 1970 (Henderson et al. 1972). Various species of fish were sampled at 50 locations throughout the United States in 1969. These sites were reexamined in the following year, along with 50 additional stations. Total mercury residues above the detection limit (0.05 mg/kg) were present in 129 of 145 samples in 1969 and in 373 of 393 samples in 1970. The median mercury level was 0.15 mg/kg for both years. Values ranged from ≤0.05 mg/kg to 1.25 mg/kg in 1969 and from <0.05 mg/kg to 1.80 mg/kg in 1970.

The data from this study indicate some general patterns. High mercury levels occurred most frequently in fish from Atlantic coastal streams and Columbia River System. Most fish containing high levels were species near the top of the food chain, a finding indicating possible biomagnification of mercury (see Biological Fate). The lowest mercury concentrations were reported in the two samples from Alaskan streams and in samples from the Colorado River System and Mississippi River tributaries in the Great Plains region (Henderson et al. 1972).

Various studies have shown that fish caught in reservoirs have higher mercury concentrations than those caught in free-flowing sections of rivers (Battelle 1977). One explanation for this phenomenon is that when mercury-rich sediments are deposited in reservoirs, these ares provide ideal conditions for bacterial formation of methylmercury (Battelle 1977). This form of mercury is readily accumulated by fish (see Biological Fate).

Data are available concerning freshwater biota in contaminated and uncontaminated areas. Price and Knight (1978) studied Lake Washington and the Sardis Reservoir, two unpolluted freshwater bodies in Mississippi. They found a mean mercury level of 0.4 mg/kg in plankton and a mean value of 0.11 mg/kg in clams. Sediment samples from this reservoir contained an average of 0.05 mg/kg mercury. The authors noted that the mercury concentration in the clams was only slightly higher than that in the sediments, a phenomenon reported in observations of clams in other relatively uncontaminated areas. Also, trophic conditions of the water may result in higher mercury levels in plankton as compared with those in clams (Price and Knight 1978). Knight and Herring (1972) studied 73 largemouth bass (*Micropterus salmoides*) in the Ross Barnett Reservoir in Mississippi. Since there are few industrial sources of mercury near this reservoir it is hypothesized that mercury in the impoundment comes from natural sources, waste items disposed of by the public and agri-

TABLE 7. CONCENTRATIONS OF MERCURY DETECTED IN AQUATIC BIOTA

Location	Concentration (ug/1)	Source	Comments
Sites throughout the United States	0.15 (median value)	Henderson *et al.* (1972)	
Lake Washington and Sardis Reservoir, Mississippi	0.4 (mean value-plankton) 0.11 (mean value-clams)	Price and Knight (1978)	Uncontaminated area; 19 samples of each species
Ross Barnett Reservoir, Mississippi	<0.05-0.74 (range)	Knight and Herring (1972)	Uncontaminated area; 73 fish
Wisconsin River	0.07-0.56 (range)	Sheffy (1978)	34 crayfish
Two Canadian Lakes	0.20-3.79 (range of means)	Moore and Sutherland (1980)	Contaminated area
Georgia (salt marsh)	0.3-9.4 (range for invertebrates) 0.1-2.4 (range for benthic organisms)	Gardner *et al.* (1978)	Contaminated area
Sites throughout the United States	0.0-6.98 (range)	NMFS (1975)	
North Atlantic Offshore Waters	0.154 (mean)	Greig *et al.* (1975)	41 species of fish
Sites in the United States, Canada and Europe	.02-.46 (range for mussels) <.01-.19 (range for herring) .08-3.85 (range for pike)	Holden (1973)	Contaminated and uncontaminated areas
California (Palos Verdes Shelf)	all mean values 0.5	Eganhouse and Young (1978)	Contaminated area
Georgia (salt marsh)	0.008-0.104 (range)	Standiford *et al.* (1973) Potter *et al.* (1975)	Area with high natural mercury levels

cultural products. The bass contained mercury levels of <0.05-0.74 mg/kg. Average mercury concentrations varied according to the weight of the fish, with the lightest fish containing an average of <0.12 mg/kg mercury and the heaviest fish containing an average of 0.45 mg/kg.

Sheffy (1978) conducted mercury analyses on 34 crayfish from the Wisconsin River and compared values found in industrialized and non-industrialized sections of the river. Mercury was present in the abdominal muscle of crayfish in concentrations of 0.07-0.56 mg/kg (wet weight), with higher mean values occurring in the southern (industrialized) section of the river. Sheffy (1978) noted, however, that the highest mercury levels were found in crayfish approximately 30 km from the industrial plants. This finding was attributed to physical characteristics of the Wisconsin River, which appear to have influenced the transport and accumulation of mercury. (See Battelle 1977)

A number of studies have focused exclusively on contaminated freshwaters and the organisms that inhabit them. Moore and Sutherland (1980) investigated two polluted Canadian lakes. Discharge of mercury-laden wastes from a gold mine into Giauque Lake was terminated in 1968, and discharges into Thompson Lake from a different gold mine were discontinued in 1949. Although mercury levels in the water of these two lakes were usually below detection limits, the sediment was contaminated. In Giauque Lake average mercury levels were 3.79 mg/kg in lake trout, 1.75 mg/kg in northern pike, and 1.22 mg/kg in round whitefish. Northern pike in Thompson Lake had mean mercury concentrations of 1.69 mg/kg and mercury values for whitefish averaged 0.20 mg/kg. Moore and Sutherland (1980) concluded that northern pike in Thompson Lake are still accumulating mercury from tailings deposited 30 years ago. They also noted that while only a small part of these lakes had been contaminated, fish throughout the lake had elevated mercury levels. Presumably this was due to movement of fish between contaminated and uncontaminated areas.

Gardner *et al.* (1978) studied a variety of organisms from a salt marsh ecosystem, which had been contaminated by discharges from a chlor-alkali plant. Mercury levels in salt marsh invertebrates ranged from 0.3 mg/kg to 9.4 mg/kg while mercury levels varied between 0.1 mg/kg and 2.4 mg/kg in benthic organisms inhabiting a river in the area.

Phillips *et al.* (1980) determined levels of mercury in fish in the Tongue River Reservoir, Montana. This area is in the vicinity of mining activity, and was investigated due to the future prospect of extensive coal mining in the area. Samples taken in 1978 showed that Northern pike had the highest concentrations of total mercury, with a maximum of 1.53 ug/g wet weight. In addition, 29% of the samples showed levels greater then 0.5 ug/g, the FDA limit in effect at that time. The authors suggested that future development of coal mining might result in increased mercury levels in fish in the area.

While mercury contamination from anthropogenic sources usually results in elevated mercury levels in aquatic biota, it should be noted that high mercury concentrations may also result from natural sources. In a study of fish in Saskatchewan waters, Sumner et al. (1972) found elevated mercury levels (0.11 mg/kg - 1.3 mg/kg) in fish from four lakes which were located far from industrial areas. The authors suggested that bedrock containing high levels of mercury might be the source of the contamination.

Mercury has been found in marine fish, as well as in freshwater species, although it has been observed that, overall, saltwater fish contain slightly lower mercury concentrations (NRC 1978, Battelle 1977). A study by Koli et al. (1978) of freshwater and saltwater fish species from South Carolina supports this conclusion.

A preliminary study by the National Marine Fisheries Service (NMFS 1975) investigated fish in coastal waters throughout the United States. Out of 106 species tested, only six had median levels greater than 0.05 mg/kg. The highest mercury concentrations were reported in fish from the southeastern coastline. Concentrations ranged from 0.0-6.98 mg/kg in East Coast fish, from 0.003-3.57 mg/kg in West Coast fish and from 0.001-1.511 mg/kg in Gulf Coast fish.

In contrast to this NMFS study, Roberts et al. (1975) report that the highest mercury levels in shellfish are found off the coast of Massachusetts and the Brunswick area of Georgia, while the lowest values were found in the Gulf of Mexico (with one exception in Texas).

A later, more comprehensive study of trace metals in aquatic biota was conducted by NMFS (Hall et al. 1978). Tissues of 204 species of fish, mollusks and crustaceans gathered from 198 sites throughout the coastal United States (including Alaska and Hawaii) were analyzed. Most fish and all crustaceans had mean mercury concentrations below 0.3 mg/kg. All mercury levels in mollusks were less than 0.1 mg/kg. Extrapolation of the data by the authors of the study indicates that less than 2% of the United States fish catch intended for consumption contains mercury levels greater than 0.5 mg/kg.

Greig et al. (1975) studied invertebrates, plankton and fish from North Atlantic offshore waters. Mercury levels in invertebrates were generally less than 0.1 mg/kg and all plankton sampled had mercury values less than 0.05 mg/kg. Forty-one species of fish were sampled and their average mercury concentration was 0.154 mg/kg. The author notes that this level is consistent with values reported in other studies. Fish with relatively high levels of mercury were the cusk (mean: 0.49 mg/kg) and the spiny dogfish (means: 0.44-0.53 mg/kg).

Marine animals have been studied in polluted, as well as unpolluted areas. An international cooperative study reported mercury levels in various species of freshwater and saltwater fish (Holden 1973). Data for mussels, herring and pike are summarized in Table 7. There was considerable overlap in concentration ranges in fish from polluted and unpolluted areas. Eganhouse and Young (1978) sampled tissues of six benthic animals living in a contaminated area near the Palos Verdes Peninsula in California. The Los Angeles County municipal wastewater outfalls were the sources of this contamination. For all samples, mean levels of total mercury were below 0.5 mg/kg while mean levels of organic mercury were below 0.05 mg/kg (wet weight). These data were compared with findings from other studies. The authors concluded that despite the contamination of the organisms' habitat, mercury levels in these animals were relatively low and similar to values found in related animals in other areas of the world.

These data indicate that marine biota located in contaminated water do not always have high mercury concentrations. These data are in contrast to studies of freshwater fish, which usually show elevated mercury levels in polluted areas.

This finding might be related to the observation (noted earlier) that overall, marine fish tend to have lower mercury values than freshwater species. Possible explanations for this phenomenon include the greater dilution and dispersion capacity of oceans and the far-ranging habits of many large marine organisms (Battelle 1977).

Various studies indicate that methylmercury comprises 61-100% of the total mercury in fish (Henderson et al. 1972, Buhler et al. 1973, Huckabee et al. 1974, Gardner et al. 1975, Knauer and Martin 1972). Gardner et al. (1978) reported that 21-100% of the total mercury present in the muscle of fish from a contaminated salt marsh was methylmercury.

Most studies of mercury in aquatic biota have focused on the threat of human exposure from mercury in seafood. However, there are some data available concerning aquatic plants and plankton.

Plants were analyzed in Lake Powell, Arizona, an area of high natural mercury levels (Standiford et al. 1973, Potter et al. 1975). Mercury concentrations ranged from 0.008 mg/kg to 0.104 mg/kg in vascular plants.

Knauer and Martin (1972) found that 12-67% of the total mercury content of phytoplankton was methylmercury. In contrast, Gardner et al. (1978) found only trace amounts of methylmercury in plants in a contaminated salt marsh in Georgia.

7. Terrestrial Biota

Mercury concentrations in terrestrial vegetation have been found to vary between different types of vegetation, and also between polluted and unpolluted areas. Table 8 summarizes these data, as well as other data presented in this section.

Mercury levels in trees in a number of unpolluted areas averaged 0.02-3.03 mg/kg (Shacklette 1970, Huckabee 1973), while averages ranged from 0.2 to 1.0 mg/kg in urban areas (Shacklette 1970, Smith 1972).

TABLE 8. MERCURY CONCENTRATIONS DETECTED IN TERRESTRIAL BIOTA

Location	Concentrations (mg/kg)	Type of Biota	Source	Comments
Unspecified	0.02-0.03 (range of mean values)	Trees	Shacklette (1970) Huckabee (1973)	Uncontaminated areas
Unspecified	0.2-1.0	Trees	Shacklette (1970) Smith (1972)	Urban areas
Tacoma, WA	1.1-4.0	Garden Vegetables	Ratsch (1974)	Near a copper smelter
Oak Ridge, TN	0.025	Grass	Huckabee (1973)	Near a stack emitting fly ash
Cades Cove, TN	0.092-0.118	Mosses	Huckabee (1973)	Unpolluted area
Florida	2.65-10.1 (range of mean values) 40.1 (high values)	Raccoon Hair	Cumbie (1975a) Cumbie and Jenkins (1974)	
Georgia	0.13-37.6	Mammal Hair	Cumbie (1975a, b) Cumbie and Jenkins (1974)	
State of Washington	0.02-11.67	Game Birds	Adley and Brown (1972)	246 birds
Jackson, Mississippi	.014-.085 (range)	Pigeons	Knight and Harvey (1974)	Values in claws

Shacklette (1970) also reported that trees growing over a cinnabar vein in Alaska contained high levels of mercury (1.0-1.25 mg/kg).

Herbaceous growth in unpolluted areas contains mercury levels similar to those found in trees in similar areas (Devendorf 1975, Standiford et al. 1973, Gay 1976). Values increase near sources of contamination. Analysis of garden vegetables growing within 3.2 km of a copper smelter in Tacoma, Washington, showed mercury concentrations of 1.1-4.0 mg/kg (Ratsch 1974).

Grasses do not appear to accumulate mercury as readily as other types of vegetation. Huckabee (1973) reported a mercury level of 0.025 mg/kg in grass near a stack emitting fly ash in Tennessee.

Studies indicate that mosses and lichens tend to accumulate more mercury than other types of vegetation. For example, mercury concentrations in mosses ranged from 0.092 to 0.118 mg/kg in Cades Cove, Tennessee (an unpolluted area), while other vegetation in the area had average mercury levels of 0.02-0.03 mg/kg (Huckabee 1973).

The far-reaching influence of anthropogenic sources of mercury on vegetation is evident in some observations in Connecticut (Mondano and Smith 1974). At distances up to 14 km from New Haven, mercury levels in trees are similar to those found in trees located in the city.

Mercury levels in mammals vary with geographical area, as well as among and within species (Battelle 1977). Cumbie (1975a, 1975b) and Cumbie and Jenkins (1974) studied mammals in the southeastern United States. Concentrations were reported for mammal hair, as mercury burdens tend to be relatively high in that part of the animal. In Florida, mean mercury values in raccoon hair ranged from 2.65 mg/kg in juveniles to 10.1 mg/kg in adults, with one report of what appears to be an unusually high mean value of 40.1 mg/kg in juvenile raccoons in Dade County. In Georgia, mean values ranged from 0.13 mg/kg for white-tailed deer in the Piedmont to 15.9 mg/kg for raccoon in the same area, and 37.6 mg/kg for otter in the Lower Coastal Plain. (All measurements are on a dry weight basis in hair.)

Feeding habits influence mercury accumulation in mammals. The highest mercury levels are found in carnivores whose diets include aquatic organisms. Herbivores tend to have lower levels than carnivores and omnivores (Battelle 1977). Lynch (1973) noticed that squirrels in rural areas had higher mercury burdens than those near cities, a situation that may have resulted from their ingestion of seeds treated with mercurial fungicides.

Mercury has been found in birds throughout the United States with residues varying between years, seasons, regions and species (Battelle 1977). However, one nationwide monitoring program for starlings found no differences in mercury levels between birds in urban and rural areas (Battelle 1977).

Clark and McLane's (1974) study of 329 woodcock from 23 eastern and midwestern states indicates a trend of higher mercury concentrations in southern woodcock. Other regional variations were noted by Heath and Hill (1974) who found mercury levels were higher in ducks from the Atlantic and Pacific flyways than in samples from the Mississippi and Central flyways.

Differences in diet may also influence mercury accumulation in birds. Heath and Hill (1974) found that black ducks had mercury burdens about twice those of the less carnivorous mallard ducks. Consumption of mercury-treated seeds may also significantly increase residues in birds (Lynch 1973).

Adley and Brown (1972) studied 246 game birds in the State of Washington. The highest mean mercury levels were 11.67 mg/kg and 0.29 mg/kg found in the livers of mergansers and teal, respectively. The authors noted that, out of all the species they studied, these two were the only ones whose diets included aquatic organisms. Other average mercury levels ranged from 0.02 mg/kg mercury in livers of grouse to 0.16 mg/kg in geese.

Knight and Harvey (1974) studied pigeons in Jackson, Mississippi to collect data on mercury levels in wild birds in urban areas. Brains of the birds contained an average of .022 mg/kg and values in claws ranged from .014 to .085 mg/kg. The authors suggested that these mercury residues may have accumulated from ingestion of treated grains and seeds, as well as from exposure to natural sources of mercury.

Some studies have shown declining mercury levels in some birds in recent years (Battelle 1977). They have attributed these reductions to declines in the use of pesticides and improved disposal practices for natural wastes.

B. ENVIRONMENTAL FATE

1. Overview

a. Methodology

This section characterizes the environmental fate of anthropogenic mercury released by processes that contribute significant quantities of the metal to the air, water and soil. The discussion emphasizes the form of mercury for each discharge, and the processes that determine its subsequent transport upon release to the environment. A general overview of the environmental chemistry of mercury produced by Versar, Inc. (1978a) has been the basis for this section. Other studies available in the literature that support the observations noted are discussed as relevant. Biological pathways have been treated separately from physicochemical and bulk transport pathways (see IV.C), although the processes promoting the biological production of methylmercury are detailed here.

b. Major Environmental Pathways

The major pathways of physical transport and relative rates at which they occur are designated in Figure 7. Atmospheric emissions (Pathway 1) have been segregated into point source and dispersive emissions. Combustion processes, such as incineration, smelting, and coal combustion, are point sources contributing to highly localized pollution; dispersive sources such as volatilization of mercury from paints and outgassing from the earth contribute to the concentration of mercury found in background levels.

Pathway 2 follows the flow of mercury originating from disposal sites for solid waste and mine tailings. As environmental controls restrain further discharges to air and water, the quantity of mercury disposed of upon land surfaces can be expected to increase. It is also the pathway for agricultural applications.

Mercury discharged with industrial process effluents into local surface waters or publicly-owned treatment works (POTW) is reviewed in Pathway 3. The fate of mercury in POTWs is described in Pathway 4.

Figure 8 gives a more general overview of all major pathways of anthropogenic mercury and the relative total contributions of the mercury-consuming activities to each environmental compartment. The major recipients are the land (mostly at specific disposal sites) and air compartments. The migration of mercury in groundwaters to nearby surface waters has not been shown in this figure, but under the proper conditions, the process can occur very rapidly. The importance of this transport pathway, however, is not well understood at this time. Also not represented in the figure is the high concentration of mercury in sediments compared with the overlying water and in soils subject to contamination by airborne mercury.

c. Important Fate Processes

In aquatic systems, mercury is concentrated in the sediments in aerobic waters, sorbed primarily onto hydrous iron and manganese oxides, clays, and organic material. The bulk of mercury transported in the water column in at least some cases is in association with the dissolved solids (Perhac 1974). The primary species are organic complexes such as with humic acid; the aqueous chloride and hydroxide are the predominant inorganic species. In anaerobic waters, the solubility of mercury decreases; under reducing conditions, mercury will be precipitated as mercury sulfide. In slightly reduced sediments, methylation of mercury results, especially in acidic waters. Most mercury species are available for methylation [upon conversion to Hg(II)], except for the insoluble HgS. Demethylating bacteria also exist, but the rate of demethylation is much slower than the rate of methylation.

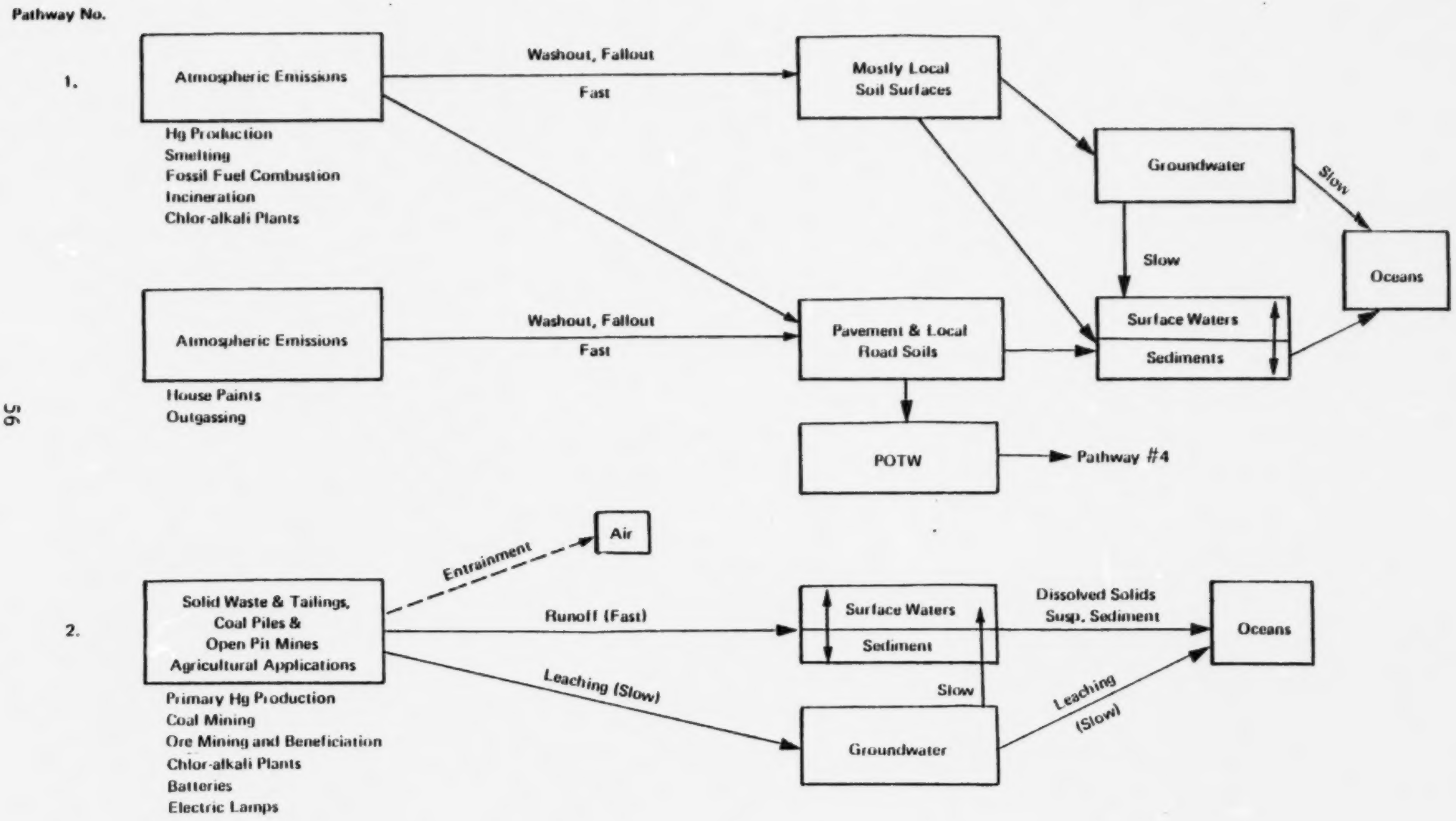


FIGURE 7 MAJOR ENVIRONMENTAL PATHWAYS OF MERCURY EMISSIONS

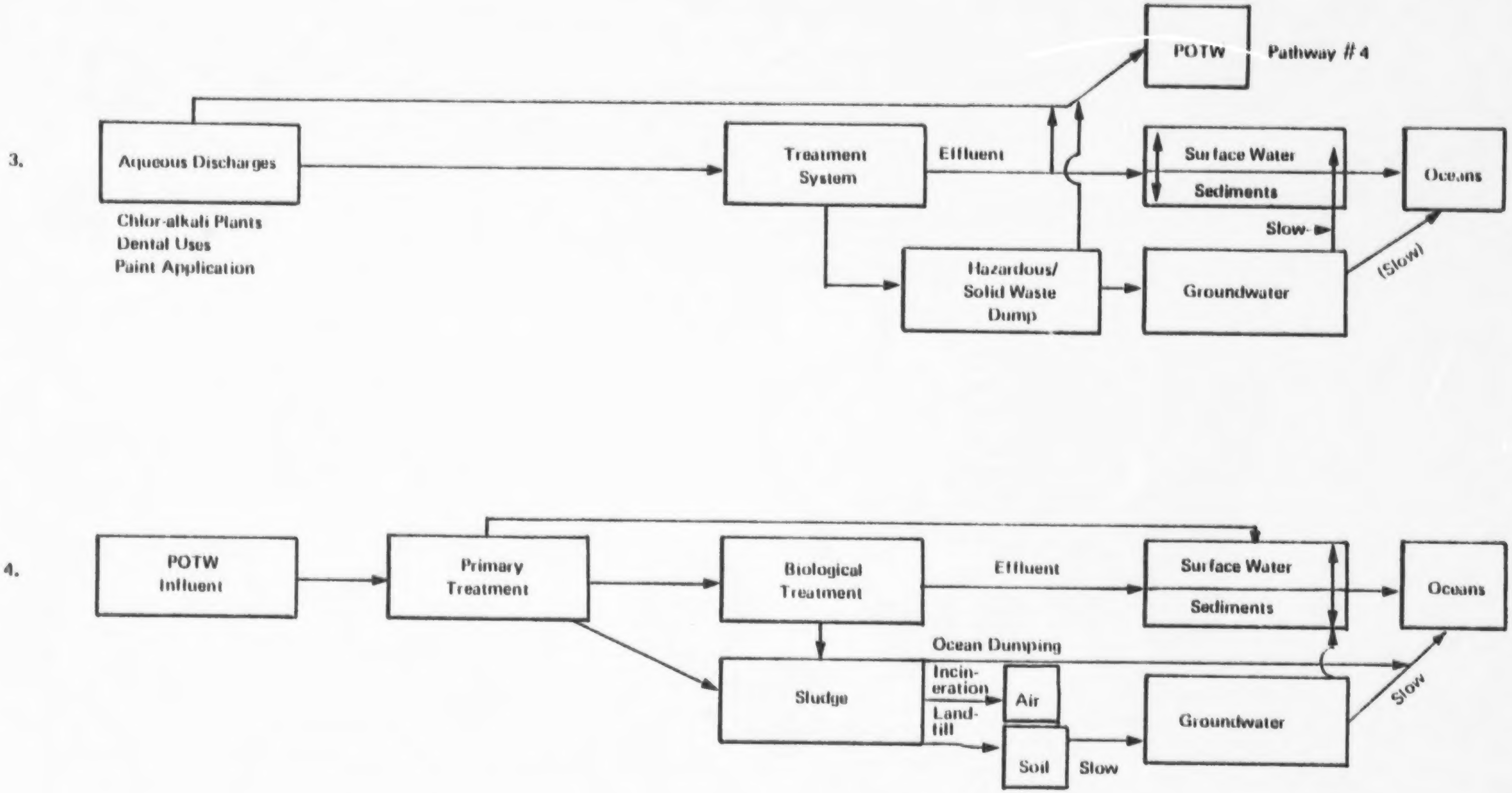
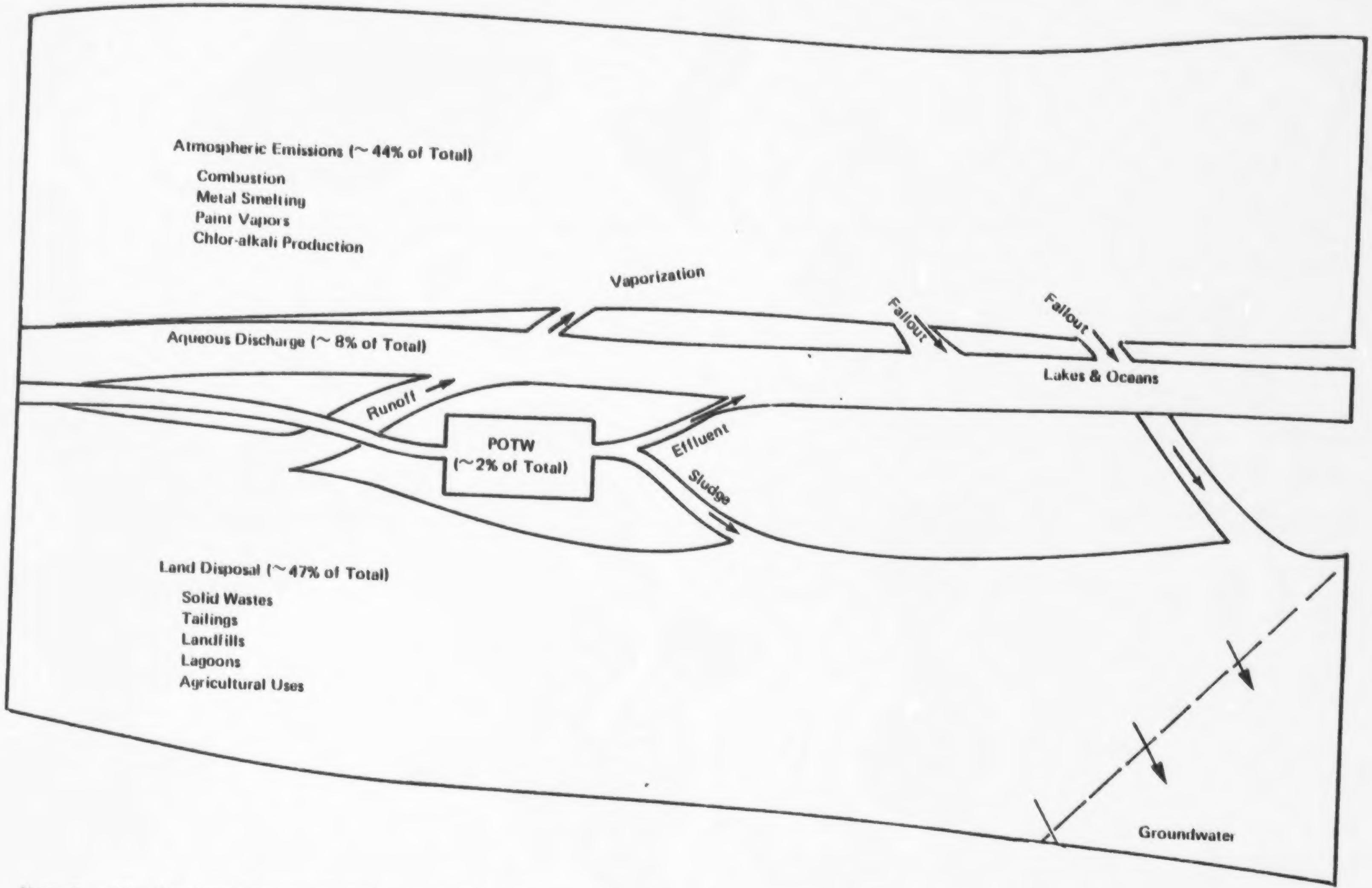


FIGURE 7 MAJOR ENVIRONMENTAL PATHWAYS OF MERCURY EMISSIONS (Continued)



Note: Quantities of anthropogenic mercury emissions moving in each pathway are roughly in proportion to the thickness of each pathway shown. The figure is derived from the materials balance of mercury described by Chapter III.

FIGURE 8 SCHEMATIC DIAGRAM OF MAJOR PATHWAYS OF ANTHROPOGENIC MERCURY RELEASED TO THE ENVIRONMENT IN THE U.S. (1979)

Atmospheric emissions of mercury consist of mercury sorbed onto submicron particulate matter and the elemental mercury vapor. Particulate mercury (about 5% of the total) is expected to be short-lived in the atmosphere; dry fallout and washout of mercury particulates contribute to mercury deposition upon local soils, urban pavements, and surface waters. Mercury, as a vapor, may be longer lived in the atmoshere, contributing to background concentrations.

Mercury is present in soils as a result of atmospheric deposition, solid waste and sludge disposal, and agricultural uses. Most of this mercury remains in the top few centimers of soil, sorbed onto organic matter, clays, and iron and manganese oxides, above a pH of about 5. The potential for translocation of mercury to the groundwater is generally small, but is greater in sandy, porous sites or in low pH environments, with a high water table. Volatilization from soils is probable, especially in dry soil containing little organic material or clay.

2. Physicochemical Pathways

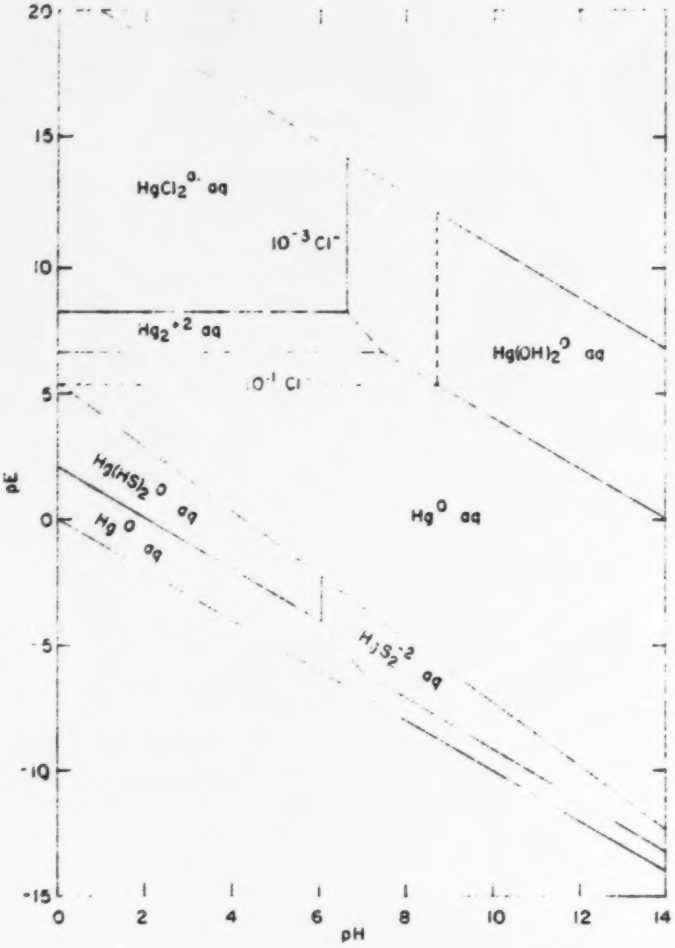
a. General Fate Discussion

i. Aqueous Complexation

The concentration of soluble mercury in water is directly related to parameters such as pH, the oxidizing potential of the water, the presence of other competing ions (e.g., calcium, magnesium and iron), the concentration of precipitating agents (e.g., OH^-, $S^=$, PO_4^{-3}, $CO_3^=$), and the concentration of complexing agents. Generally, at low pH values, and in low alkalinity waters, mercury will be more soluble; at high pH levels, and in high alkalinity waters, mercury is usually found complexed with organic ligands, chlorides, and hydroxides. In natural aerated waters, mercuric mercury [Hg (II)] is the stable form. Less soluble forms of mercury typically found in aerated natural water include the oxide (.0053 g/100 g); in anaerobic waters, the insoluble sulfide predominates. Figure 9 illustrates the presence of aqueous mercury species as a function of pH and pE (Rubin 1974).

ii. Mercury Transport in Aqueous Systems

Mercury distribution and transport in river systems have been researched by Rubin (1974), Khalid et al. (1975), and the Ottawa River Project Group (1979). Mercury in the water column is concentrated by inorganic hydrous oxides, clays, and organic suspended solids to approximately 5-25 times the concentration found in the water (Rubin 1974). The work of Perhac (1974), which dealt with metals other than mercury, shows that, although most metals are concentrated in suspended sediments, the ratio of the mass of suspended sediments to the mass of water is so low that metals are transported in most cases as dissolved solids. This observation is supported for mercury by the work of the ORPG (1979), which determined the distribution of mercury in bed sediments, suspended solids, and water. Table 9 summarizes their



Note: 25°C and 1 atm total pressure. Solution contains 10^{-3} *M* $SO_4^{=}$ and 10^{-3} or 10^{-1} *M* Cl⁻ . Dashed line represents expanded field boundary of $HgCl_2$. High solubilities exist over the upper one-third and extreme lower right of the diagram.

Source: Rubin (1974)

FIGURE 9 STABILITY FIELDS OF Hg AQUEOUS SPECIES AS A FUNCTION OF pE AND pH

TABLE 9. DISTRIBUTION OF MERCURY IN THE OTTAWA RIVER

Component	Total Hg (ug/kg)	Annual Flow of Mercury Through Systems (kg)
Water	.03	1.3×10^3
Suspended Solids	440[1]	3.4
Bed Sediment (4cm)	410[1]	2.9×10^2

[1] Dry weight

Source: Ottawa River Project Group (1979).

findings, which confirm that dissolved mercury in the water column is the principal species transported.

Mercury is more easily adsorbed than most other metals. Figure 10 shows that although less easily sorbed than copper, mercury is adsorbed more readily than nickel, cobalt, zinc and cadmium (Vuceta and Morgan 1978). Mercury adsorption onto sediments is strongly influenced by the redox potential and pH and the sediment characteristics. Khalid *et al.* (1975) determined that mercury added as $HgCl_2$ to Mississippi River sediment is adsorbed at high pH (6.5-8.0) levels and reducing conditions. The pH dependence of adsorption is seen by the following data generated for an invariant redox potential: at pH 5, 50-75% of the added mercury remained soluble, at pH 6.5, the percentage was 1-13% at pH 8.0, less than 0.5% remained in solution (Khalid *et al.* 1975).

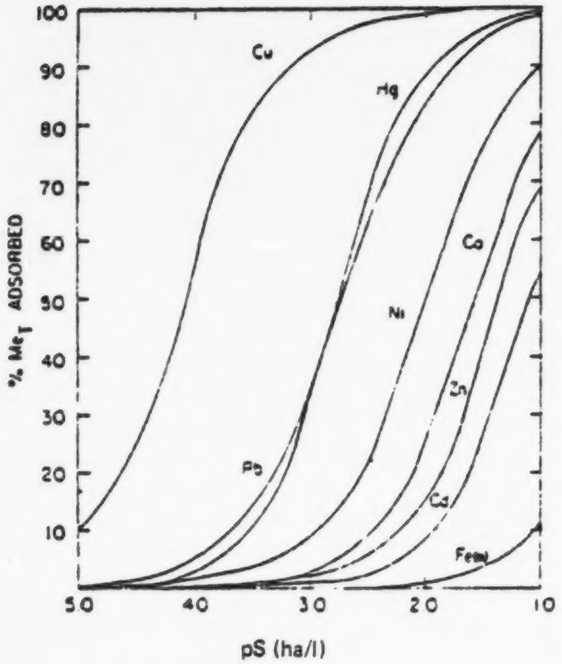
Desorption is also determined by pH and redox potential. For all pH levels, desorption is negligible in reduced waters. In aerated waters, the rate of desorption increases inversely with pH.

The Ottawa River Project Group (1979) determined that the mercury concentration in sediment of a localized section of a river channel fluctuated according to the organic fraction in the sediment. Mercury concentrations ranged from 0.05 mg/kg for pure sand to 1.8 mg/kg for organic material. These authors also inferred a mercury half-life in the sediment of 1-2.5 years, on the basis of mercury concentrations monitored for 3 years. The clearance mechanisms cited were sediment transport, desorption and transport, bioaccumulation, volatilization, or "removal" created by burial from new sediments.

iii. Atmosphere

Mercury is released as a vapor or aerosol into the atmosphere. The aerosol fraction reported is about only 5% of the total mercury in air (WHO 1976). Vapor species encountered in an analysis of air in Florida were distributed as follows: 25% Hg(II), 21% CH_3Hg^+, 49% Hg(0), and 1% $(CH_3)_2Hg$ (Johnson and Braman 1974). Since the concentration of mercury in the atmosphere depends upon the temperature, concentrations tend to be higher during the summer than in the winter (Krenkel 1973). Versar (1978a) estimates that a mean residence time for mercury in the atmosphere is 4-11 days. Transformation of organic mercury compounds and elemental mercury, which can occur during this period, are promoted by UV light for the former group and oxidation to the divalent ion in the presence of water for the latter (WHO 1976).

Mechanisms for removal of mercury from the atmosphere include wet and dry deposition. Some dispute has arisen as the the efficiency of wet deposition in the removal of mercury from the atmosphere. Krenkel (1973) cites several examples of near 100% removal of mercury during a rainfall. Van Horn (1975), on the other hand, cites examples in which the particulate mercury is removed, but the remaining vaporized mercury component (~90% of total) remains in the atmosphere despite rain and snowfall.



Note: pH = 7, $p\epsilon$ = 12, $p_{CO_2} = 10^{-3.5}$ atm, pC_r = 4.16

Source: Vuceta and Morgan (1978)

FIGURE 10 **ADSORPTION OF TRACE METALS IN OXIDIZING FRESH WATERS AS A FUNCTION OF SURFACE AREA OF SiO_2 (s)**

The form of mercury in rainwater is not known (NRC 1977), but it may be an adsorbed species on particulate matter or the divalent ion, resulting from the oxidation of elemental mercury or methylmercury particularly if the rain is acid in nature. The divalent and methylmercury forms are available for further translocation within the air, soil and water.

iv. Soils

The fate of mercury in soils follow three routes: volatilization, leaching, and conversion to methylmercury. Rogers (1978) determined the relative volatilization rates of various mercury species applied to sandy, loam, and clay soils. The most soluble mercury species [$HgCl_2$, $Hg(NO_3)_2$, and $Hg(CH_3H_3O_2)_2$] disappeared from soil more quickly than the less soluble, HgO, and insoluble HgS. The volatility rate was progressively lower in sand, loam, and clay soils. Table 10 summarizes some of the data Rogers (1978) generated for soils saturated at 50% of field capacity, at pH 8-9.

Mercury transport via soil solution is dependent upon soil pH, soil content, microbial activity, and the species of mercury present. Inorganic mercury species [Hg(0) and HgS] undergo principally oxidation-reduction reactions; Hg(0) to the divalent ion and HgS to soluble sulphates or sulfites in the presence of oxygen. The divalent ion is capable of complexing and chelating to organic matter. Organic mercury species, such as those used in fungicides, are very unstable, and are transformed in acid soils to the divalent ion (WHO 1976).

The extent of inorganic mercury adsorption is related directly to the organic and clay content in soils. Versar (1978b) reports that mercury has an affinity for the sulfhydryl groups in organic matter, and montmorillonite and illite clays. Soil horizon profiles indicate that soils rich in clay and organic matter do not permit significant translocation of mercury. At low pH levels, however, some of these species are solubilized and translocated.

v. Methylation

Biological: Mercury may undergo biological methylation under both aerobic and anaerobic conditions in water and soil solution. In anaerobic, mildly reducing systems, mercury, as the divalent ion, reacts with methylated vitamin B-12 (CH_3 B-12) to form methyl and dimethylmercury. Microbes in the environment that are dependent upon CH_3 B-12 are capable of methylating inorganic divalent mercury. It is important to note that the form of mercury must be the divalent inorganic species for methylation to occur. Most mercury compounds introduced by anthropogenic releases into the environment are eventually transformed to Hg (II). Some examples are elemental mercury, phenyl mercuries, alkyl mercuries, and alkoxy-alkyl mercury (NRC 1977). Excluded from this list is HgS, which due to its extremely low solubility and the prevailing anaerobic conditions, is not available for methylation (Lexmond *et al.* 1976).

TABLE 10. PERCENTAGE OF MERCURY EVOLVED FROM SOIL IN 144 HOURS

	% Removed in Type of Soil		
Hg	Sand	Loam	Clay
$HgCl_2$	33.8	32.9	14.2
HgO	19.6	15.0	6.4
HgS	0.2	0.3	0.2

Source: Rogers (1978).

Aerobic and facultative anaerobic bacterial species recognized as being capable of methylating mercury are *Klebsiella pneumoniae*, *Escherichia coli*, and *Clostridium cochlearum*. The amount of mono- versus dimethyl mercury formed is a function of initial mercury concentrations, and the pH of the system (D'Itri 1972). Low initial concentrations of Hg (II) and neutral to alkaline waters favor formation of dimethyl mercury. The low water solubility and the high vapor pressure of this species result in rapid volatilization from the system. Monomethyl mercury may be returned to the system in acid rains, although methylmercury has not been detected in rain (WHO 1976).

Higher initial concentrations of mercury and pH levels less than neutral, promote CH_3Hg^+ formation. A slight degree of acidity pushes the mono-di-methlymercury equilibrium toward the formation of CH_3Hg^+ (D'Itri 1972).

Berdicevsky *et al.* (1979) researched the formation of methylmercury in sterile and unsterile marine sediments under both aerobic and anaerobic conditions. Mercuric chloride was used as the initial mercury species. No methylmercury was produced in the sterile medium. Methylmercury production was also shown under aerobic conditions. The anaerobic cultures produced methylmercury in amounts that varied inversely with the initial concentration and decreased over time, unlike the trend noted for aerobes. Losses of methylmercury were attributed to evaporation and adsorption onto the experimental glassware. Table 11 shows production of methylmercury as a function of initial concentration and time.

Abiological Methylation: Rogers (1978) found that mercury could be methylated in sterile soils. He isolated a substance as something belonging to the low molecular weight fraction of soil organic matter, which he concluded promoted methylation. The conversion rate from inorganic to methylmercury was directly proportional to increasing temperature, decreasing pH (at pH levels greater than 5), and increasing concentration of mercury ion. Of the clay, loam, and sand soils tested, clay had the greatest ability to methylate mercury, followed by loam and then sand.

Demethylation: Methylmercury may be decomposed abiotically or with the aid of microbes. Chemical demethylation results from photolytic decomposition of methylmercury-sulfur complexes, which are the principal form of environmentally available methlymercury species. The reaction sequence is shown below (Lexmond 1976):

$$\begin{array}{lcl} CH_3HgS & & CH_3\cdot + HgS \\ & \xrightarrow[\text{light}]{\text{UV}} & \\ CH_3HgSR & & CH_3\cdot + SR\cdot + Hg(0) \end{array}$$

TABLE 11. METHYLMERCURY FORMATION OVER TIME AND RELATIONSHIP WITH CONCENTRATIONS OF MERCURY IN ANAEROBIC CULTURES

Starting	Methylmercury Levels (as Hg)					
Total Hg	Day 2		Day 5		Day 12	
ug/ml	ug	%Total Hg	ug	% Total Hg	ug	% Total Hg
0.1	77.3	77.3	98.0	98.0	5.2	5.2
3.1	n.d[1]	n.d	97.9	3.1	2.6	.08
10.0	n.d	n.d	23.3	0.2	2.4	.02
30.1	n.d	n.d	20.4	0.1	2.4	.01

---

[1] n.d. = not detected

Source: Berdicevsky *et al.* (1979).

Methylmercury is also chemically decomposed when the mono-dimethylmercury equilibrium is shifted toward production of dimethylmercury under some conditions such as in slightly alkaline waters.

In both aerobic and anaerobic waters microbial demethylation is accomplished by several bacterial species capable of forming elemental mercury and methane from methylmercury. Shariat et al. (1979) found that 21 of 40 bacterial strains isolated from soil, sewage, and sediments were able to demethylate mercury. The organisms are believed to have developed a resistance to methlymercury poisoning by evolving enzymes capable of hydrolyzing the methyl-carbon bond, and reducing the Hg (II) produced to Hg(0) (NRC 1977). The high volatility of Hg(0) permits escape of mercury from the soil or water system. The rate of demethylation is reported to be several orders of magnitude slower than methylation. In the presence of demethylating organisms, however, steady-state concentration of CH_3Hg^+ is lower than it would be otherwise (NRC 1977).

Summary Statement: The concentration and speciation of soluble mercury in the water column is dependent upon the pH and redox potential of the water and the nature of complexing ligands. In natural aerated waters, Hg (II) is complexed with organic ligands, chlorides and hydroxides. In reduced environments, mercury will be present as mercurous oxide and HgS.

Mercury adsorbs onto iron and manganese oxides, clays, and organic matter in the sediments. Its tendency to adsorb exceeds that of all other divalent metals, with the exception of copper. Suspended solids concentrate mercury; this concentration may exceed that of the sediments due to a greater number of adsorption sites on suspended sediment. However, the amount of suspended sediments is usually small enough that the major quantity of mercury in water bodies is found in the sediments and water column as the dissolved solid. Methylation of mercury occurs in slightly reduced, anaerobic and to a lesser extent, aerobic sediments. Biological methylation proceeds at a rate faster than biological demethylation. Acidic waters favor the formation of monomethyl mercury, while neutral to alkaline waters favor production of water-insoluble dimethylmercury.

In soils, mercury is adsorbed above a pH of 5; organic ligands, especially humic acid and clays enhance adsorption. Compared with other metals (excepting copper), mercury demonstrates the greatest tendency to adsorb. In acid environments, mercury will be available in the soil solution, although to a lesser extent than other metals.

b. Atmospheric Transport

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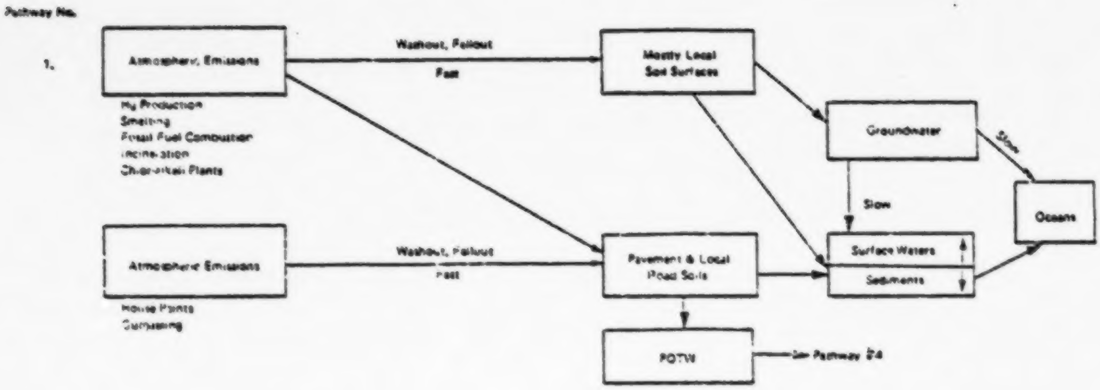
i. Overview



Anthropogenic releases of mercury to the atmosphere result from a number of point and dispersive sources. The primary point source emissions are due to thermal processes that release mercury as the elemental vapor or sub-micron aerosol (<4.5 um). Principal thermal sources are solid waste and sludge incineration, chlor-alkali production plants, fossil-fueled power plants, and metal smelters (such as copper smelters). Dispersive sources include such processes as volatilization of paints containing and outgassing from the earth (see Chapter III).

Thermal processes release mercury as a vapor or as a sub-micron aerosol. The elemental mercury vapor tends to be concentrated and adsorbed onto particulate matter in the atmosphere. This sorptive process is often cited as the reason for 100% removal of mercury by rainfall via washout of particulates. As discussed above, only about 5% of the total mercury resides on the particulate fraction in the atmosphere. Versar (1979) cites a mean residence time in air of 4-11 days.

Deposition of mercury in urban areas was not specifically documented in the literature available, although mercury levels in urban runoff suggest that it is occurring. Particulate washout will deposit mercury from local point sources on pavement, where it will be transported to a POTW or local surface waters. The behavior of mercury in surface waters will be similar to that detailed in the general chemical fate section.

The available literature also did not document incidences of atmospheric fallout of mercury into surface waters. Due to the tendency of mercury to vaporize and be widely dispersed, washout and dry fallout should contribute to mercury concentrations in the oceans. Vaporization and re-entrainment of aerosols from the water surface will continue the atmospheric mercury cycle.

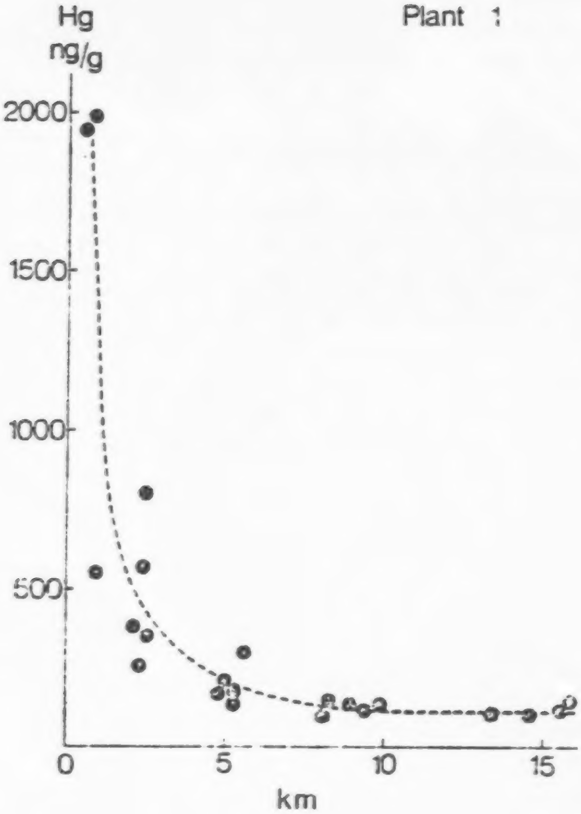
ii. Municipal Solid Waste and Sewage Incineration

Soldano et al. (1975) conducted a survey of airborne mercury emissions from sewage treatment plant incinerators in order to determine the transport differences of organomercury and elemental mercury. The survey indicated that the concentration of inorganic mercury [as Hg(II)] decreased as a function of distance from the source, whereas the concentration of monomethyl mercuric chloride (CH_3HgCl) increased with distance. The reasons proposed for this observation were (1) that the alkyl mercury species comprise the major fraction of mercury emanating from the plant, and (2) that the high volatility of this particular species would allow for more rapid transport upon release than would the inorganic fraction.

In another study concerning municipal solid waste incinerators, Law and Gordon (1979) analyzed the combustible and non-combustible portions of the waste to ascertain which fraction contributed significant metal releases to the atmosphere. The combustible portion of solid waste was thought to contain most of the mercury. Actual measurements were not reported for mercury, as they were for other metals. Negligibly low quantities of mercury were thought to be in the flyash and fine bottom ash remains of the incinerated waste; no mercury was accounted for by the non-combustible fraction of the waste.

iii. Chlor-Alkali Plants

Airborne emissions of mercury from chlor-alkali plants have been studied extensively in Sweden and Canada. Pollution control devices have curtailed mercury emissions to the air by more than 90% (Flewelling 1971). The fallout pattern of these emissions has been studied as reflected in concentrations of mercury in moss (Wallin 1976) and snow (Jernelov and Wallin 1973). Figure 11 illustrates the decrease in mercury concentrations in moss as a function of distance from a chlor-alkali plant. The same trend was observed in snow. The greatest concentrations of mercury are seen within 1 km of the plant, as a result of wet and dry deposition and sedimentation. The quantity of mercury accumulated by the moss was only 20% of that reported for snow. Reasons postulated for these observations are (1) that mercury fallout is a temperature-dependent



Source: Wallin (1976)

FIGURE 11 CONCENTRATION OF MERCURY IN MOSS SAMPLES AS A FUNCTION OF THE DISTANCE FROM A CHLOR–ALKALI PLANT IN SWEDEN

process and proceeds more rapidly in cold weather, and (2) concentrations reported for moss are complicated by tissue decomposition, absorption efficiency of the moss, and direct water losses to the ground through the moss mat. However, the important conclusions of both of these studies are that the highest concentrations of mercury are found close to the chlor-alkali plant emission stack, but surprisingly that this deposition represents only a small percentage of the total plant emissions. Dilution to background concentrations in fact appears to be the fate of most of the airborne mercury.

iv. Coal and Other Fossil Fuel Combustion

Mercury emissions from coal combustion are significant, not because of concentrations of mercury existing in coal, but because of the enormous quantities of coal used to generate power. Coal combustion is estimated to contribute about 93 MT of mercury to the atmosphere each year, with other fossil fuels releasing 84 MT of mercury (see Chapter III).

Billings and Matson (1972) studied a series of coal samples containing an average of 0.3 mg/kg Hg, and found that approximately 95% of the mercury is released with the flue gas. The fate of the mercury emissions from and deposition near a coal-fired power plant was studied at Four Corners, New Mexico, and the findings differ from concentrations normally reported for mercury in soil close to a combustion point source (Crockett and Kinnison 1979). The mean mercury concentrations in soil (ng/g) samples obtained at sites around the plant in concentric circles with radii measuring 1.0, 2.9, 6.8, 15 and 30 km, were 22, 16, 14, 15, 13, and 16 ng/g, respectively. Thus, it would appear that mercury levels above background are found at distances of 1 km or less from the plant.

v. Metallurgical Plants

Zinc and copper deposits can contain about 100-300 mg/kg Hg (Habashi 1978), which is released during smelting as a vapor associated with SO_2 gas. Prior to the implementation of pollution controls, the mercury was released directly to the atmosphere. Since then, sulfur dioxide scrubbers concentrate approximately one-half of those mercury releases (Habashi 1978).

vi. House Paints

Mercury is present in water-based house paints as a bactericide and a fungicide. It can apparently volatize quite rapidly from painted surfaces. On the tenth day following application to an indoor surface, the indoor concentration was 1000 times the exterior ambient air level of mercury. Elemental mercury and phenyl mercuric acetate, with small amounts of methyl mercuric chloride were detected (U.S. EPA 1976).

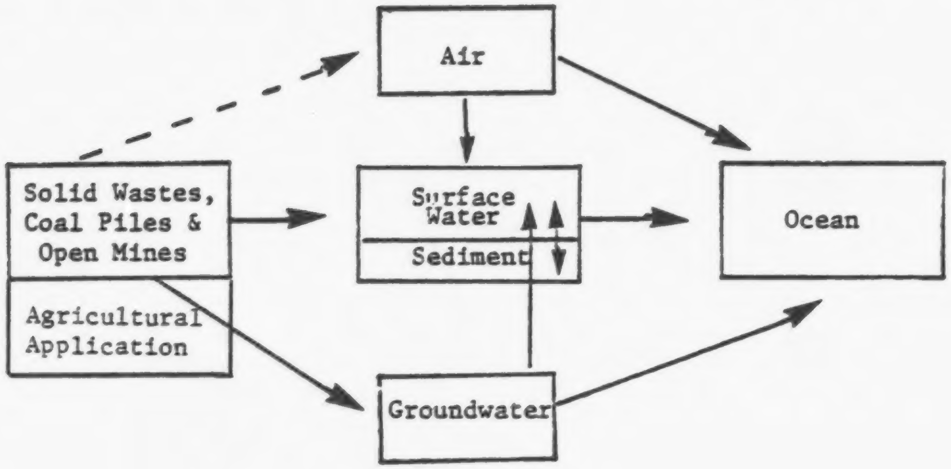
vii. Summary Statement

Mercury enters the atmosphere from point source combustion processes and from widely dispersed sources from which vaporization occurs. Mercury is sorbed onto sub-micron particulates, which have a residence time in the atmosphere that is subject to meteorological conditions such as washout and fallout. Localized pollution of soils, pavements and surface waters results from point source emissions. Mercury as the vapor will be longer lived in the atmosphere, eventually contributing to background concentrations.

c. Solid Wastes and Agricultural Applications

i. Overview

Pathway 2



Most of the mercury-containing solid wastes arise from mineral ore processing and coal mining or from municipal or hazardous wastes. Solid waste from mining operations results from the overburden of surface mining, and the low-grade portions of mineral-ore deposits. The tailings, which contain highly concentrated minerals, are produced as a final waste product of mineral concentration operations (Martin and Mills 1976). Mercury is also released to land during landfilling and lagooning of industrial and municipal sludges; flyash disposal; and the abondonment of mercury-containing products such as batteries, scientific instrumentation. and paint. Another major source of mercury released to land is the application of mercury for agricultural purposes.

The oceans or lakes that are fed by streams or groundwater from mined areas, solid and hazardous waste sites, or agricultural areas, may serve as the ultimate sinks for mercury released from solid waste. However, these sites are probably themselves the ultimate sink for mercury, in the form of the insoluble sulfide or sorbed tightly onto clay minerals and organic matter.

ii. Mine Tailings and Coal Piles

Mercury was mined in both surface and underground mines, which in the United States exist in the West, primarily in Nevada and California. The principal ore of mercury is cinnabar, HgS, which is very insoluble and stable. The amount of mercury lost during mining and primary production is reportedly minimal. The tailings produced contain, on the average, 5 mg/kg of mercury (Van Horn 1975). The tailings and waste produced since 1850 are estimated to be total 60 million MT in the U.S. (Martin and Mills 1976). Mercury is also associated with other sulfide-rich ores such as copper, zinc, and coal. Localized leaching of mercury from these mineral ores and coal tailings does not appear to present a problem, as will be discussed below (Van Horn 1975).

iii. Acid-Mine Discharge

Leachate from controlled coal piles and mine tailings contains low concentrations of mercury. Acid mine drainage from abandoned mines solubilizes metals and aids in their transport. Acid mine drainage results from the exposure of fine particulates to air, which oxidizes the metal sulfides (e.g., HgS) to sulfuric acid. The impact of acid mine drainage and extent of metal transport within streams depend upon the buffering capacity (alkalinity) of the stream. Letterman and Mitsch (1978) studied the impact of acid mine drainage emanating from several abandoned coal mines in Pennsylvania. One discharge tested was typical of concentrated acid mine discharge (pH 2.6, alkalinity as $CaCO_3$ = 0 mg/l); another was closer to neutral (ph 6.0) and buffered by contact with the limestone strata underneath the mine. In both cases the mercury concentration in the leachate was less than 0.0003 mg/l. The low concentrations of mercury may be due to (1) a low concentration of mercury originally present in the coal (which is probable since it is an Eastern coal), (2) adsorption of the mercury within the coal pile, or (3) volatilization.

Martin and Mills (1976) in their studies of the problems associated with abandoned mines did not feel that mercury presented a problem. In cases where high levels of mercury in the sediment were reported, they were thought to be due to discharges occurring when the mine was active.

The principal fate of mercury discharged to local streams with acid mine drainage is probably adsorption to suspended particulates in the water column, with subsequent deposition in the stream bed, or quiescent lake system. Methylation is a probable occurrence over time, releasing mercury for biotic uptake and transport via volatilization.

iv. Solid Waste Disposal Sites

Mercury losses from municipal waste are principally released to the land. The main contributors to mercury in solid waste are batteries, control instruments, lamps, and tubes and switches (Van Horn 1975). Losses of mercury via leachate from properly designed landfills have not been documented (Van Horn 1975).

Roulier (1975) reports on two studies of metal transport with landfill leachate. In the first study, leachate collected under anaerobic conditions from municipal refuse was passed through columns of well-characterized soils. The concentration of mercury in the leachate was below the detection limit of 0.0005 mg/l.

The second study was a more realistic simulation of a properly-designed landfill. Leachate was collected anaerobically from two operating landfills and passed through columns packed with clay and quartz sand at a rate of 2 pore volumes per month. The initial mercury concentrations in the landfill leachates were 0.2 mg/l and 0.0008 mg/l. The results demonstrated that 96.8% of the mercury was attenuated by the column. The principal mechanism responsible for the attenuation was attributed to precipitation (the pH was neutral to alkaline). The researchers of this study concluded that clay-lined landfills will provide suitable preventive measures against metal transport by landfill leachate (Roulier 1975).

The conclusion to be drawn from both of these studies is that mercury migration to groundwater is probable in poorly operated landfill sites. Van Horn (1975) found that over one-half of the landfill sites operating at the time of this study did not comply with regulatory requirements. A properly operated site, on the other hand, should not release mercury to the environment.

v. Flyash Disposal Ponds

Only minimal mercury is translocated from flyash disposal ponds (Theis et al. 1978). Groundwater from wells surrounding the ponds consistently contained mercury levels below 0.2 ug/l.

vi. Agricultural Applications

Mercury has been applied to agricultural fields as a fungicide seed dressing, insecticidal foliar spray, and as a minor constituent of fertilizer and sludge used for the purposes of soil amendment. Alkyl mercurials are no longer used for fungicidal or insecticidal uses due to the problems associated with methylmercury. Phenyl mercurial compounds are now used most frequently. Mercury in fertilizer results from reuse of the sulphuric acid originally used in SO_2 scrubbers, which concentrate mercury vapor along with the SO_2 gas (Habashi 1978).

Translocation of mercury applied in agriculture within the soil profile has been cited in a literature review by Krenkel (1973). In soil profiles of rice paddies and orchard fields, the mercury concentration profile was found to be a direct function of the clay fraction and type in the soil. One soil, containing insignificant mercury concentrations, despite 10 years of continual application, was found to be underlaid by a gravelly sand, which permitted loss of mercury by leaching. A soil containing higher mercury concentrations was found to contain a large fraction of montmorillonite clay. In subsequent studies of the adsorption tendencies of phenyl mercuric acetate (PMA) and $HgCl_2$, adsorption on clays decreased according to the type of clay and in the following order: montmorillonite > allophane > kaolinite. Adsorption was greatest at pH 6. Van Horn (1975) states that PMA and other phenyl mercurials are not immobilized in the surface soil layer, and are easily leachable. The PMA remaining in the soil (approximately 50% of that applied) is also subject to loss via vaporization. Mercury is also subject to loss from the soil surface by erosion and runoff. Mercury in this case will be transported as an adsorbed species to surface waters.

Little information was available concerning mercury contamination through sludge and fertilizer application to agricultural sites. One can assume that mercury in the sludge will be in a form less available for biological uptake and leaching. Accumulation in the soil surface is likely for mercury applied in this form.

vii. Summary Statement

Solid wastes, coal piles, and tailings are point sources of mercury disposed of on land. Of these sources, mercury exposed as a result of mining practices is potentially subject to greater translocation in the environment due to the acid nature of the leachate, but there is no evidence of such movement occurring.

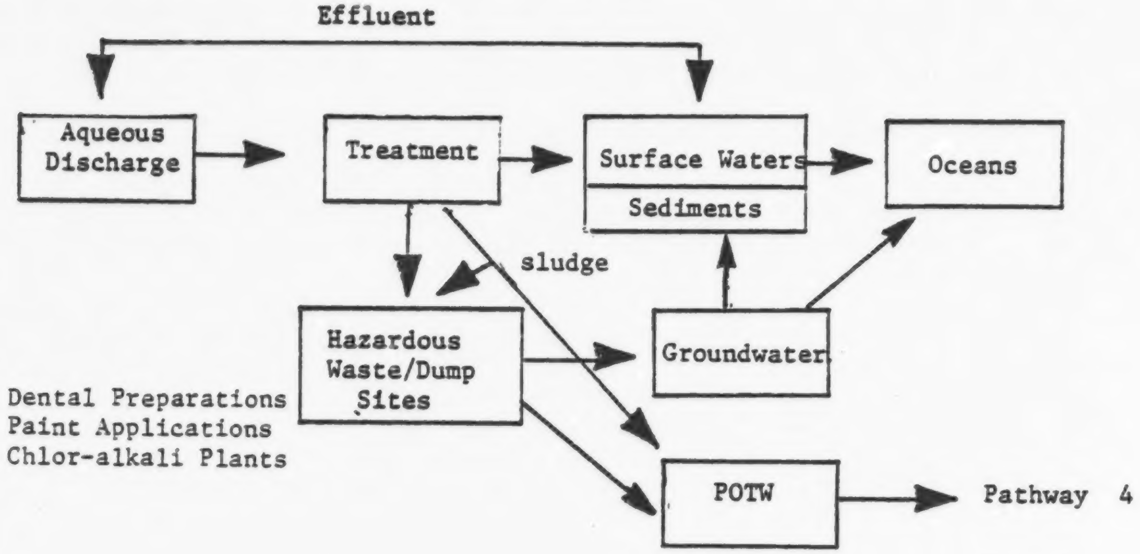
Studies of municipal waste landfills have revealed mercury concentrations in leachate ranging from 0.0005 mg/l to .2 mg/l. Mercury is quickly attenuated by the soil, provided a high clay content exists and the pH is alkaline. No data were found regarding groundwater contamination, though such contamination should not occur in a properly operated landfill disposal site.

Mercury used in agricultural settings can be translocated through leaching or volatilization. The importance of the former pathway is dependent upon the clay content of the soil. Erosion and consequent runoff are likely to be important pathways for mercury used for agricultural purposes.

d. Aqueous Industrial Discharge

i. Sources and Treatment

Pathway 3



Pathway 3, shown above, considers the fate of mercury discharged with industrial wastewater effluents. The industries and uses that contribute to these discharges are dental preparations, paint applications, and use of electrical apparatus. The effluents from these sources are discharged with or without treatment into natural waters or municipal wastewater treatment systems. Waters discharged to the latter are treated in Pathway 4.

The quantity of mercury discharged from chlor-alkali plants and paper and pulp industries has been slight since regulations were imposed on these industries in 1970. No mercurials have been used in Canada's paper and pulp industry since 1970 (Paavila 1971), and the abatement measures have reduced mercury emissions from Canada's chlor-alkali plants by 99% (Flewelling 1971). Mercury releases from these industries have also been reduced in the U.S.

ii. Distribution in Surface Waters

The fate of mercury discharged as an industrial process effluent was not well described in the available literature. Krenkel (1973) concluded that mercury is concentrated in the sediment below outfalls from chlor-alkali plants.

Cooke and Beitel (1971) have performed a mass balance on mercury entering the Great Lakes, partially from chlor-alkali plants. According to their calculations, if mercury released to the Great Lakes from chlor-alkali plants were eliminated after 1970, then the mercury entering the watershed per year would be reduced from 2.2 million lb per year to 1.5 million lb. They attribute some of the remaining mercury discharges to urban runoff. Other sources considered were losses from ore reduction, fuel consumption, laboratory use, agriculture, dental uses, and disposal of manufactured products. Figure 12 illustrates the predicted concentrations of dissolved mercury in the Great Lakes as a result of mercury discharges from chlor-alkali plants.

Jackson (1979) analyzed the mercury concentrations in the sediments of two lakes, the first of which is fed by a river receiving paper and pulp and chlor-alkali plant discharges, and the second of which is fed by the outflow of the first lake. The ratio of mercury to organic carbon for the first lake was significantly greater than that for the second lake.

Similar ratios for four other metals did not vary between the two lakes, a finding suggesting that concentrations of these metals resulted from normal weathering and erosion of the watershed. In the contrast. the results for mercury clearly implied that the first lake was acting as a sink for mercury introduced into the river upstream. Mercury reaching the sediment would be subject to methylation as described previously.

iii. Sludge Disposal

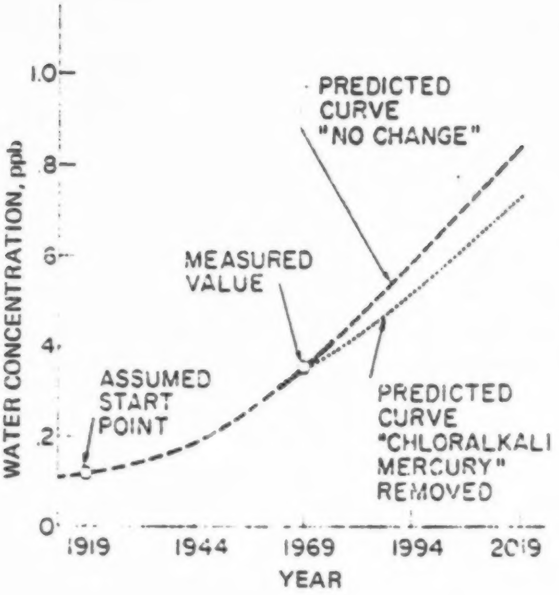
The sludge generated by industrial effluent treatment is normally disposed of in a solid or hazardous waste dump, or a settling pond. A properly designed hazardous waste dump should prevent further translocation of mercury due to leaching. At some sites, the leachate is collected and sent to a POTW (with or without further treatment). Groundwater contamination is possible in a poorly operated landfill or settling pond. The speed with which mercury is translocated in this pathway is fairly fast in soils of low organic matter and clay. The fate of mercury in solid waste sites was reviewed in Pathway 2.

iv. Ultimate Sinks

The major sinks for mercury associated with treated industrial effluents are, in the short term, hazardous waste dumps, settling ponds, or sites used for the disposal of sludge generated by POTWs. The long-term sinks, as discussed earlier, are the oceans and lake sediments.

v. Summary Statement

A major fraction of the mercury in aqueous industrial discharges appears to be concentrated in the sediments in the vicinity of the



Source: Cooke *et al.* (1971).

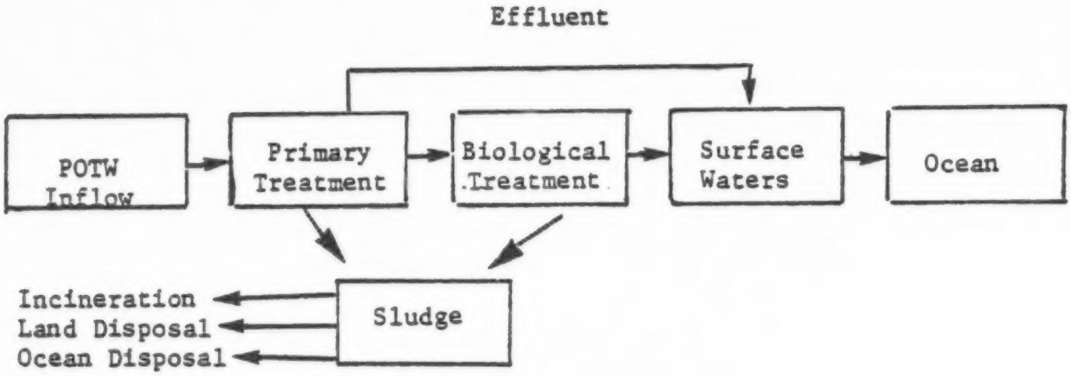
FIGURE 12 PREDICTED VALUES OF THE AVERAGE CONCENTRATION OF MERCURY DISSOLVED IN THE LOWER GREAT LAKES

source. It is distributed principally with the organic, sulfide, and clay components of the sediments, which are subject to methylation. Disposal of sludge generated by waste treatment in a properly-operated landfill should prevent further translocation of mercury.

e. POTW

i. Treatment Schemes

Pathway 4



Pathway 4, shown above, describes the fate of mercury in wastewaters that are introduced into a Publicly-Owned Treatment Works (POTW). The inflow to the POTW may consist of combinations of industrial and commercial effluents, domestic wastes, and runoff. Though the nature of the influent is consequently quite varied, typical concentrations of mercury in the influent will be about .0004 mg/l (Levins *et al.* 1979).

The degree to which mercury is removed from the raw wastewaters, and thus the concentration of mercury in the discharged wastewaters and sludges, depends on the type of treatment involved. Levins *et al.* (1979) report that average removal efficiencies of treatment processes are 37.2% for primary treatment and 58.4% for secondary. Oliver and Cosgrove (1974) report mercury concentrations in the effluent following primary and secondary treatment as follows:

	Hg Concentration (mg/l)	
Treatment Stage	Total	Dissolved
Raw Sewage	.007	.0006
Primary Effluent	.003	.00003
Secondary Effluent	.001	.00005

The efficiency of biological treatment systems in removing large slugs of mercury has been studied by Neufeld and Hermann (1975) for aerobic sludge digestors, and by Lingle and Hermann (1975) for anaerobic systems. In the latter study, phenyl mercuric chloride and mercuric chloride were introduced into a simulation of an anaerobic sludge digestor at concentrations of up to 2,200 mg Hg/l. For both the mercury species and all concentrations, about 96% of the mercury was partitioned into the sludge solids, with 4% remaining with the sludge supernatant, on suspended solids greater than 0.45 um. The authors also determined that the largest concentration of mercury (2,200 mg/l) inhibited digestion, while 1,560 mg/l did not. Analysis for methlymercury produced during digestion revealed negative results.

The study by Neufeld and Hermann (1975) of aerobic sludge digestors determined that mercury (added in concentrations of up to 1000 mg/l) reached almost complete equilibrium and was nearly all removed (95%) by the biological floc within 3 hours. Toxicity studies revealed that aerobic treatment is inhibited temporarily at concentrations equal to or greater than 2.5-5.0 mg/l $Hg^{\cdot 2+}$ (Ghosh and Zugger 1973). The biological floc becomes acclimated to larger doses within a few hours.

Conclusions contrary to those indicated by the proceding two studies are seen in the work of Mytelka _et al_. (1973) on treatment plant efficiencies as obtained from a survey of POTWs in New Jersey, New York and Connecticut. In this study, 90% of the plants surveyed had influent mercury concentrations of 0.0052 mg/l entering an aerobic digestor, and 0.0050 mg/l exiting in the effluent. This implies that very little of the mercury is partitioned in the sludge portion, although initial concentrations were quite low. The authors use these findings as support for recommendating mandated pre-treatment of waste effluents by industries prior to discharge into the sewers.

ii. Sludge Disposal

Sludge disposed of on land may go to a sanitary landfill, or be spread for the purpose of amending the soil. The form of mercury in sludge has not been revealed in this literature search, but it is known that the metal remains bound to the organic matter of the sludge and is converted into an insoluble state (Oliver and Cosgrove 1974). Thus, disposal of the sludge in sanitary landfill sites should not create major problems since the potential for leaching into the groundwater is minimized by the form of mercury in sludge.

Sludge that is incinerated will contribute close to 100% of its mercury content to the atmosphere. More detailed information can be found in the description of atmospheric emissions (Pathway 1).

iii. Surface Water Discharge

The behavior of mercury discharged with POTW effluents into local waters will be similar to that described for aqueous pathways (Pathway 3).

Morel *et al*. (1975) used a chemical equilibrium model to trace the fate of metals present in sewage upon discharge in the ocean. The sewage used in this model was in the reduced state, so mercury was present as the sulfide. The model predicts that dilution and oxidation will solubilize the sulfide to the mercuric ion. This transformation will occur some distance from the outfall and mercury will reach concentrations similar to background levels.

The validity of this model is supported by the work of Eganhouse *et al*. (1978) who studied the distribution and speciation of mercury from a sewage outfall in Palos Verdes. They found that mercury near the diffusers was inorganic in nature, probably the sulfide, whereas moving away from the outfall, increasing concentrations of mercury were associated with organics. Schell and Nevissi (1977) and Whaling *et al.* (1977) found that the intertidal organisms -- *Ulva fuscus*, mussels, and clams -- tended to reveal increased uptake near the outfall, but the results were within sample variability. The second study of North Carolina estuaries showed slight mercury elevations between control and discharge estuaries in the roots of *Spartina alterniflora* (0.13-0.14 mg/kg). Mercury concentrations were increased in snails toward the outfall, while they were not in the small sample of oysters analyzed. The authors concluded that the current practices for effluent disposal into the estuaries of North Carolina were acceptable to the ecology of those systems.

iv. Summary Statement

The concentration of mercury in POTW influent averages about 0.4 ug/l. The effectiveness of its removal appears to be very high for biological treatment processes, in which mercury is partitioned into the sludge portion of the waste. Sludge spread for the purposes of soil amendment is not likely to enhance the solubility or mobility of mercury. In municipal landfills, the concentration of mercury in leachate ranged from 0.0005 mg/l to 0.2 mg/l. Mercury is expected to be quickly adsorbed in soils containing clays and organic matter. Mercury in aqueous effluents is principally sorbed onto suspended solids. Discharges to marine systems can result in solubilization of mercury due to oxidation and dilution. In fresh waters, mercury is expected to be partitioned into the sediments, or be associated with dissolved solids in the water column.

C. BIOLOGICAL FATE

1. Introduction

Mercury is commonly found in the tissues of biota, especially in aquatic species. The following section describes the fate of mercury in biota and discusses:

(1) The significance of the form of mercury, environmental parameters, and route of exposure on rate of uptake;

(2) Half-lives and bioconcentration factors in biota;

(3) Biomagnification in trophic levels; and

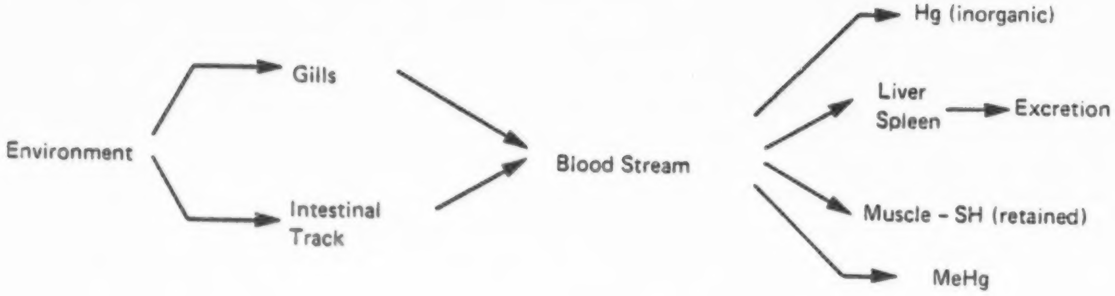
(4) Bioaccumulation in terrestrial ecosystems.

2. Uptake of Mercury

Most mercury found in fish tissue is in the form of methylmercury (Uthe *et al.* 1973, Hildebrand *et al.* 1976, Phillips and Russo 1978). Although *in vitro* liver preparations have been reported to methylate inorganic mercury (Matsumara *et al.* 1975), most researchers attribute methylmercury in tissues to direct uptake of that form of the metal (Phillips and Russo 1978). Methylmercury is rarely detected in the water column even directly above methylating sediments, an observation that is attributed to immediate biotic uptake of the newly formed compound (WHO, 1976). DeFreitas *et al.* (1974) found in fish that the methyl form of mercury was taken up 100 times as rapidly as the inorganic form from water and five times as rapidly from food. Other differences between the behavior of the two forms are discussed below.

Methylmercury is absorbed very efficiently through biological membranes. Gut absorption efficiency in fish is 90% for methylmercury and 15% for the inorganic form (Norstrom *et al.* 1976). The methyl form is excreted more slowly (Miettinen *et al.* 1976). Once inside the body, most of the methylmercury quickly becomes bound to sulfhydryl groups in protein in a non-diffusible form (WHO, 1976). Figure 13 schematically describes the pathway of mercury in a finfish. Within muscle tissue, mercury has a greater affinity for myofibrin and sacroplasmic protein than for non-protein nitrogenous compounds and insoluble muscle residues (Arima and Umemoto 1976).

The variables affecting the rate of mercury uptake include temperature, pH, and mercury concentration in water. Findings on such effects have been reported for several species and are assumed, at this time, to be applicable to fish in general. The rate of uptake conforms to zero-order kinetics during the initial uptake phase, with a linear relationship to water temperature (Hartung 1976). Cember *et al.* (1978) found a 0.066 exponential increase in rate of uptake per degree increase in temperature between 9°C and 33°C in bluegills. This is attributable to the increased pumping of water over the gills as a function of an increase in metabolic rate with temperature rise (Burkett 1974). Burkett (1974) found the temperature dependence to drop off above a temperature of 21°C, an observation suggesting interference by the substance at that point reducing efficiency of membrane transfer. Mercury uptake (of mercuric chloride) increased as pH decreased, especially below pH 7.0



Source: Windom *et al.* (1976)

FIGURE 13 APPARENT MERCURY PATHWAYS IN FINFISH

(Tsai et al. 1975). The concentration of mercury in water was important to uptake kinetics in fathead minnows (Olson et al. 1975) and rainbow trout (McKim et al. 1976). Greater bioconcentration factors were observed at the higher water concentrations.

3. Bioconcentration

Bioconcentration factors (BCF)[1] for mercury in biota commonly range from two to five orders of magnitude over water levels. Table 12 presents examples of BCFs reported for laboratory and field studies in aquatic systems. Laboratory-measured BCFs are commonly greater than values measured in natural systems (Burkett 1974). Invertebrates tend to exhibit the highest BCFs of aquatic species, on the order of 10^6 (see Battelle 1977).

In a field study in the Ottawa River Project (ORPG 1979), concentrations in various species ranged from three to four orders of magnitude above water conconcentrations (see Table 13). The organic fraction of the total mercury content varied by species, ranging from 0.3 to 0.85 and highest in fish. Due to differences in absorption efficiency in the gut (90% for methylmercury, 15% for inorganic) (Norstrom et al. 1976), higher organic concentrations were expected in upper trophic level species, which would be exposed to potentially higher levels of methylmercury in their prey. Lower trophic level organisms would be exposed to low levels in water and sediment.

Mercury (both total and methyl) tends to be concentrated in the muscle, heart, liver, and kidneys of fish, based on observations on five species of fish (Bishop and Neary 1977). Considerably lower concentrations were measured in skin, scales, and bone. A negative correlation was found between mercury levels and fat content in bottom feeders and no observable correlation was found for other species.

Fromm (1977), however, found the gill in rainbow trout to be a more important site of accumulation than the gastro-intestinal tract for both methyl and inorganic mercury. The inorganic form tended to be bound to the gill mucus, however, and was less likely to enter the body. Nearly 50% of mercuric chloride in two species of fish was found to be associated with external mucus (Tsai et al. 1975).

4. Route of Exposure

The routes of exposure of aquatic organisms to mercury have been a matter of some controversy. As discussed above, it is generally thought that the source of organic mercury in aquatic organisms is due to the presence of low levels of that form in the water. However, ingestion of mercury may also be an important exposure route, in addition to gill absorption. Table 14 describes these three interrelated hypotheses.

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[1] The ratio of the concentration in biota tissue to the concentration in water.

TABLE 12. BIOCONCENTRATION FACTORS FOR AQUATIC SPECIES

Species	Concentration in Water (mg/l)	Concentration in Biota (mg/kg wet wgt)	Approximate BCF	Source
Marine Plants	3×10^{-5}	3×10^{-2}	10^3	Thompson *et al*. (1972)
Marine Mollusks Crustaceans	3×10^{-5}	1×10^0	10^5	Thompson *et al*. (1972)
Marine Fish	3×10^{-5}	5×10^{-2}	10^3	Thompson *et al*. (1972)
Freshwater Plants	1×10^{-4}	1×10^{-1}	10^3	Thompson *et al*. (1972)
Freshwater Inverts	1×10^{-4}	1×10^1	10^5	Thompson *et al*. (1972)
Freshwater Fish	1×10^{-4}	1×10^{-1}	10^3	Thompson *et al*. (1972)
Barnacle[1]	9×10^{-3} (sediment = 5×10^{-1})	3×10^{-1}	10^2	Guthrie *et al*. (1979)
Crab[1]	"	5×10^{-3}	10^1	Guthrie *et al*. (1979)
Oyster[1]	"	7×10^{-2}	10^1	Guthrie *et al*. (1979)
Clam[1]	"	1×10^{-1}	10^2	Guthrie *et al*. (1979)
Polychaeta[1]	"	1×10^{-1}	10^2	Guthrie *et al*. (1979)
Rice Fish Eggs (*Oryzias latipes*)[2,3]	3×10^{-2}	6×10^1	10^3	Heisinger and Green (1975)
Pike	-	-	10^3	Johnels *et al*. (1967)
Pike	3×10^{-2}	3×10^0	10^2	Hannerz (1968)

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[1] Field Study.

[2] Exposed to mercuric chloride.

[3] Japanese species.

TABLE 13. MERCURY DISTRIBUTION IN OTTAWA RIVER ECOSYSTEM

Component	Total Hg[1] Conc. ug/l ug/kg)	Fraction Organic	Approximate BCF
Water	~0.03		-
Bed Sediment	41	0.01	10^3
Suspended Solids	440	~0.3	10^4
Benthic Invertebrates	220	~0.3	10^4
Higher Plants	100	0.20	10^4
Fish	180	0.85	10^4

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[1] Wet wgt for biota.

Source: Ottawa River Project Group (1979).

TABLE 14. OBSERVATIONS REGARDING ROUTE OF EXPOSURE OF AQUATIC BIOTA TO MERCURY

- Organic mercury concentrations in water are usually either extremely low or not detectable.

- Usually >90% of the total mercury content found in fish and shellfish tissue is in the methyl form (NRC 1978).

Hypothesis 1 - Fish take up inorganic mercury and convert it to the methyl form in vivo

Supporting Evidence

- Inorganic mercury is accumulated by fish to concentration factors of 1 to 50 (NRC 1978).

- Evidence of *in vitro* conversion of inorganic to methylmercury (Matsumara *et al*. 1975).

Counter Evidence

- Fish experimentally exposed only to mercuric ion exhibit primarily the inorganic form in their tissue (Hannerz 1968, Cox *et al*. 1975).

- Uptake of methyl form is much more efficient than of inorganic mercury.

- Evidence of *in vivo* conversion of methylmercury to inorganic mercury in fish (Sharpe *et al*. 1977), which would counteract methylation process.

Hypothesis 2 - Methylmercury in water is the source of the methylmercury found in fish and is directly taken up through gill absorption

Supporting Evidence

- Although methylmercury is often non-detectable it is found to comprise 10-30% of the mercury found in water (ORPG 1979). In addition, current analytical methods are not sensitive enough to detect methylmercury at the concentrations at which it normally occurs.

- Gill uptake of methylmercury is extremely efficient and rapid and the compound's biological half-life is long; both factors lead to high bioaccumulation.

TABLE 14. OBSERVATIONS REGARDING ROUTE OF EXPOSURE OF AQUATIC BIOTA TO MERCURY (Continued)

Counter Evidence

- Methylmercury concentrations in water are often too low to account for tissue levels.

Hypothesis 3 - Methylmercury is transferred primarily through the food chain, entering it via bottom feeders ingesting contaminated bacteria and invertebrates associated with bottom sediment

Supporting Evidence

- Methylation of mercury takes place primarily in upper layer of sediment and methylmercury concentrations there are relatively high compared with concentrations in other aquatic compartments.

- Uptake efficiency of methylmercury from food is high, with an associated 80% efficiency as compared to a 12% efficiency for gill uptake for some fish (Norstrom *et al*. 1976).

- Studies show that higher trophic-level species associated with the water column accumulate more methylmercury from food (60% of total) than bottom feeders (25% of total), indicating significance of diet as a mercury source to species not associated with sediment.

Counter Evidence

- Does not explain extremely high concentrations in low trophic-level invertebrates not associated with sediment.

- Puts too much emphasis on benthic population as prime component of total aquatic food chain; doesn't account for producers such as algae.

Hypothesis 1 is the weakest and, alone, cannot justify the high methylmercury levels found in fish. Existing laboratory observations on methylation in higher organisms are limited and do not indicate a fast conversion rate.

Hypotheses 2 and 3 are more popular in the literature (NRC 1978, ORPG 1979) and together contribute most significantly to methylmercury levels in fish. Speculations abound as to which of the two exposure routes is more significant. In yellow perch, 80% of methylmercury in food and 12% in water passing over the gills is taken up (Norstrom _et al._ 1976). Results of research by various investigators (Terhaar et al. 1977, Suzuki and Hatanaka 1974) conclude that ingestion in fish is the more significant uptake route.

On the other hand, other researchers (Fagerstrom and Asell 1976) claim that uptake from water is more significant. These authors, however, assumed that northern pike assimilated 15% methylmercury in the diet, and 100% passing over respiratory surfaces. Phillips and Buhler (1978) found that rainbow trout assimilated 10-12% of methylmercury passing over the gills, and northern pike assimilated 15-20% of the methylmercury they ingested (Phillips 1978).

The position of the fish in the food chain may influence the relative contribution of each pathway; for the upper trophic-level species such as pike, 60% of its body burden of mercury was attributed to uptake from food, while for bottom feeders only 25% was believed to result from ingestion (Jernelov and Lann 1971, Olson _et al._ 1973), even though absorption efficiencies were similar in the two species.

Phillips _et al._ (1980) have reviewed this point recently. They concluded that Norstrom _et al._ (1976) have correctly assumed a 12% efficiency for respiratory methylmercury absorption, and that Fagerstrom and Asell (1973) appropriately assumed a 14% efficiency for dietary absorption. However, planktivores accumulate most of their methylmercury body burden from water, and piscivores derive methylmercury from both diet and water.

Using a pollutant accumulation model[3] developed by Norstrom _et al._ (1976) for uptake of mercury from water and assuming the efficiency of 20-40% for gill absorption of methlymercury, the following calculations were made (ORPG 1979). At a methylmercury concentration of 0.004 ug/l in water, net uptake of 2 ug at one week for a 1-kg fish was estimated. If a uniform distribution in the body is assumed, this would result in a tissue concentration at one week of 2.0 ug/kg and a resulting concentration ratio of 5 x 10^2. This value is compatible with observed values (see Chapter IV-B). Therefore, accepting the assumptions of this model, a very low methylmercury concentration in water can contribute significantly to typical tissue concentrations, and this supports Hypothesis 2 above.

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[3] The model uses pollutant biokinetics and fish energetics taking into account a growth dependent metabolic rate, a gill uptake pollutant based on respiratory rate, ingestion uptake based on caloric requirements (by age class) times an efficiency of absorption and excretion based on body weight multiplied by a tissue turnover rate coefficient. See publication for more detail.

The significance of food as a source of methlymercury can be illustrated by the following example. Assuming DeFreitas et al.'s (1977) gut-absorption efficiency of 90% for methylmercury and 15% for inorganic, the following equation (ORPG 1979) predicts the organic mercury fraction in food assimilated and, therefore, in the predator's body:

$$f^* = \frac{0.90}{0.75 + \frac{0.15}{f}}$$

where f = organic mercury fraction in food (unassimilated)

f^* = organic mercury fraction in predator.

Therefore, assuming retention of the mercury ingested, invertebrates could achieve a 30% organic mercury fraction from a 7% organic fraction in sediment ingested, fish a 72% fraction from ingesting invertebrates, and higher trophic level fish 94% from ingesting fish with a 72% fraction. It is important to remember that the total amount of mercury associated with biota in an aquatic system is small relative to the mass contained in the water and sediment compartments (ORPG 1979). Table 15 shows the mercury (total) distribution in the Ottawa River in the summer of 1973. The mass in biota is six orders of magnitude lower than the mass in water and eight orders of magnitude lower than in bottom sediment. Therefore, the amount taken up by biota is small relative to the total mercury load in the system.

5. Elimination

Elimination of mercury from tissue occurs very slowly. Only after 2 years in a mercury-free pond were mercury levels in yellow perch (Perca flavescens) and rock bass (Ambloplites rupestris) reduced, and the loss was attributed to tissue dilution through growth (Laarman et al. 1976). Freshwater clams (Anodonta grandis) retained methylmercury, but not inorganic forms, after transfer to clean waters (Smith et al. 1975). A longer retention time for methylmercury has also been reported in guppies (Kramer and Neidhart 1975).

Table 16 illustrates the variability in biological half-lives reported for methylmercury in various species. Half-life may vary by organ as found in the freshwater clam (Unio) (Renzoni and Bacci 1976). In addition, there is evidence that biological half-lives are temperature dependent; residence time was shorter at higher temperatures in oysters and bacteria (Cunningham and Tripp 1975a, Hamdy and Prabhu 1978). Methylmercury, as suggested earlier, has a longer half-life in biota than do inorganic or other organic forms of mercury (Miettinen 1976).

TABLE 15. DISTRIBUTION OF MERCURY MASS IN THE OTTAWA RIVER

Component	Instantaneous Mass of Mercury Present in 4.8 km Test Segment	
	Inorg (g)	Org (g)
Water	350	—
Bed Sediment[1]	13,530	135
Suspended Solids	98	29
Biota[2]	10	2.9

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[1] Sediments measured to 4 cm deep.

[2] Biota include benthic invertebrates, macrophytes, and fish.

Source: ORPG (1979).

TABLE 16. BIOLOGICAL HALF-LIVES OF METHYLMERCURY IN VARIOUS SPECIES

Species	Biological Half-Life (days)
Mouse	7
Monkey/Man	70
Seal	500
Fish:	
Flounder (*Pleuronectes flesus*)	700 - 1200[1]
Pike (*Esox lucius*)	640 - 780[1]
Eel (*Anguilla vulgaris*)	910 - 1030[1]
Rainbow Trout	346
Mussel (*Pseudanodonta complanata*)	100 - 400
Bacteria[2] (*Bacillus licheniformis*)	7 - 12

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[1] Dependent on route of exposure

[2] From Hamdy and Prabhu (1977).

Sources: WHO (1976), Miettinen (1975).

6. Biomagnification in the Food Chain

Information on biomagnification of mercury in the upper trophic levels is conflicting. A major cause of this is that field measurements of residues in different trophic levels are usually of total mercury, not methylmercury (see Battelle 1977). On the other hand, methylmercury, being more prevalent and persistent in biota, is more likely to be transferred in a food chain. Total mercury measurements indicate no magnification in higher trophic levels, while measurements of organic mercury do indicate bioaccumulation (ORPG 1979).* The mean level in predators has been estimated to be 15 times the level in primary consumers (see Battelle 1977). Table 17 shows biomagnification (according to the arithmetic mean) of mercury (presumably total) in an aquatic food chain. In a model food chain study, however, Hamdy and Prabhu (1979) found inorganic mercury accumulation in bacteria and mosquito larvae, but no magnification of inorganic or organic mercury in guppies and their predators, cichlids. Species variability in uptake rate and biological half-lives must be taken into account before biomagnification of mercury can be understood.

Fish-eating birds -- such as herons, ducks and gulls -- are often found to have mercury concentrations in their tissues and feathers, both in North America (Hoffman and Curnow 1973, Vermeer and Armstrong 1972, Hough and Zabik 1972, Stendell _et al_. 1976, Dustman _et al_. 1972, Adley and Brown 1972) and in Scandinavia (Berg _et al_. 1966, Sarka _et al_. 1978, Holt 1969, Karppanen _et al_. 1970). Concentrations have been detected as high as 23 mg/kg in muscle tissue, 175 mg/kg in liver, and 65 mg/kg in feathers (see NRC 1978). Residue concentrations in ducks have exceeded guideline levels at times (see NRC 1978). A more detailed discussion of mercury accumulations in birds can be found in the NRC's report on mercury (NRC 1978).

7. Terrestrial Biological Fate

Most terrestrial plants are able to concentrate at least small amounts of mercury (NRC 1978). The chemical form of mercury and the soil characteristics affect uptake. Elemental mercury and alkylmercuric compounds are more readily taken up by plants than the ionic inorganic form (Dolar _et al_. 1971). Alkoxyalkyl- and phenylmercury compounds are not taken up as efficiently by plants or are more rapidly degraded to inorganic mercury than is methylmercury; this led to the elimination of the latter as a fungicide in Sweden (WHO 1976). In aerated soils, the rapid accumulation of gaseous mercury results in residues of 0.2-10 mg/kg (dry weight) in plants grown in soils where gaseous mercury is released by decaying sulfides. In reducing soils where the mercury present is bound to soil constituents, typical plant concentrations are an order of magnitude lower (Kothny 1973).

*Increasing concentrations of organic mercury while total mercury remains the same suggests that concentrations of other forms of mercury are decreasing.

TABLE 17. BIOLOGICAL MAGNIFICATION OF MERCURY IN THE AQUATIC FOOD CHAIN

Organisms	No. Samples	Concentration (mg/kg) Range	Concentration (mg/kg) Arithmetic Mean	More Numerous Organisms
Algae eaters	39	0.01-01.8	0.05	Zooplankton, snails, mayfly nymphs
Zooplankton eaters	9	0.01-0.07	0.04	Insect larvae, minnows
Omnivores	9	0.04-1.16	0.45	Insect larvae and adults, scuds
Detritus eaters	12	0.13-0.59	0.54	Worms, clams, insect larvae
Predators	25	0.01-5.82	0.73	Insect larvae and adults, frogs

Source: Bligh (1971).

Terrestrial plants accumulate mercury from three sources: from mercury fungicide treatment of seeds, from foliar application of phenylmercuric sprays, and from mercury contaminated soils via root uptake. The first two sources primarily affect food crops, while the third is the most likely source of mercury residues in wild plants. Since mercury use as pesticide is extremely limited, it is expected that the direct contamination of plants, including food crops, would be limited.

Root uptake of mercury into plants from soil is most important in the vicininty of mercury sources. Natural background levels in soil exist on the order of 0.01-1.0 mg/kg, averaging 0.071 mg/kg in the U.S. (NRC 1978). In mineralized areas soil concentrations may be as high as 500 mg/kg (NRC 1978). The behavior of mercury on reaching the soil will affect plant uptake: if volatilized it is most likely to be absorbed by roots, if converted to mercuric sulfide or an organic mercury compound, it is less likely to be (NRC 1978).

Bull et al. (1977) studied the effects of proximity to a chlor-alkali plant on the mercury content of topsoils and various organisms. In the case to topsoils, grass (Festuca rubra), and earthworms (Lumbricus terrestris), mercury residues in specimens collected within 0.5 km of the plant were 30-40 times higher than specimens taken 10-30 km away. However, the authors did not clarify, in the case of the grass (F. rubra), whether the mercury was deposited on the leaf surfaces or whether the mercury was actually absorbed into the tissues. A range 8-13% of the mercury in the earthworms was in the methylated form; the organic fraction was not determined for other media. Woodmice (Apodemus sylvaticus) and bank voles (Clethrionomys glaredus) collected near the works had significantly greater concentrations of total mercury in the liver, kidney, brain, and hair than control animals. No greater than 10% of the total mercury in the rodents was in the methyl form. Since no methylmercury was known to be used in the area, the authors attributed its presence in biota to methylation of the inorganic form in soil; no mention was made of possible methylation by the organisms in which the compounds were measured.

Soils surrounding a mercury mine in Nevada also proved to supply quantities of mercury for uptake in three plant species (Gay 1976). In samples of Bromus rubens, Spharalcea ambigua, and Boraginaceae sp. collected in November and December (during dormancy), no significant residues were detected. When specimens collected during the growing season (in May) were examined, mercury was found in the range of 2.5-10 ug/kg. The developing seeds of B. rubens in particular concentrated mercury to relatively high levels. Unfortunately, the author failed to report the residues found during the winter, and did not analyze the mercury content of the soil. Consequently, no uptake rates or concentration factors were determined by this study.

Gardner et al. (1978) examined a variety of species for elevated mercury residues in a salt marsh near a chlor-alkali chemical plant. Concentrations in the roots of the marsh grass, Spartina alterniflora, reflected the variations in the mercury content of the surrounding surface sediments, suggesting that uptake is related to substrate concentrations. With the exception of the specimens from one collection site, however, other plant tissues did not have high mercury levels.

In hydroponic solutions, various crop species rapidly accumulated methylmercury hydroxide (MMH) as high as three orders of magnitude greater than the 0.006 mg/l MMH in solution (Lipsey 1972). These conditions would be relatively conducive to uptake compared with uptake from soil.

In a microcosm study, less than 1% of the total mercury applied to soil ended up in plants (Huckabee and Blaylock 1974); most remained bound to soil. Plant concentrations were not available. The insignificance of plant uptake has been supported by field studies, in which very low residues where found in plants grown in mercury-treated soils (Matti *et al.* 1975, Smart 1968, Blanton *et al.* 1975). Concentrations of mercury in plants grown in well-aerated soils apparently range from less than 0.1 mg/kg to 0.7 mg/kg (wet weight), regardless of soil mercury concentration (see NRC 1978).

D. Summary

1. Monitoring Data

Mercury has been detected in all components of the environment, including water, sediment, rocks and soils, the atmosphere, and terrestrial and aquatic biota. Elevated levels often result from anthropogenic sources, and occasionally from natural sources.

Mercury levels in uncontaminated water are generally low (.04-0.3 ug/l) and are similar for freshwater and saltwater. Values of up to about 20 ug/l mercury have been reported for water in contaminated areas. It is likely that 10-20% of mercury in water is in the form of methylmercury.

Mercury concentrations in sediment are generally higher than those in water. Levels range from ~0.05 mg/kg in unpolluted areas to over 2.0 mg/kg near industrial sources of contamination. Methylmercury generally represents no more than 1% of the mercury in sediment.

Rocks and uncontaminated soils contain similar levels of mercury. Values generally range from 0.20 mg/kg - 0.15 mg/kg, with concentrations of up to 250 mg/kg reported for sites near natural mercury deposits.

Atmospheric mercury is primarily a vapor rather than adsorbed on particulates, and is usually in the elemental form. Background concentrations range from 1 ng/m^3-5 ng/m^3 while urban levels vary from 2-60 ng/m^3. High values result from sources of contamination such as incinerators and power plants. Automobile exhaust may also contribute to atmospheric mercury pollution.

Many data are available concerning mercury in aquatic biota. Freshwater fish usually have slightly higher mercury levels than do marine fish. Most saltwater organisms contain mercury levels below 0.3 mg/kg. Values for freshwater fish generally range between <0.05 mg/kg and 1.80 mg/kg. Mercury contamination from anthropogenic sources usually is the cause of elevated mercury levels in freshwater fish; concentrations in marine organisms are less likely to be affected.

Terrestrial biota also contain detectable levels of mercury. Trees and herbaceous growth in unpolluted areas have concentrations ranging from 0.02 mg/kg to 0.03 mg/kg, with levels up to 1.25 mg/kg in areas contaminated by anthropogenic or natural sources of mercury. Levels in birds and mammals vary depending on such parameters as species, and geographical region. Feeding habits can also influence mercury accumulation in mammals and birds.

2. Environmental Fate

Mercury in the water column is concentrated on suspended solids and in sediments. Methylation of mercury is promoted both biologically and abiologically in low pH environments, and under slightly reducing conditions. In the atmosphere, most of the mercury (>90%) occurs as a vapor, while the remainder exists adsorbed to sub-micron particulate matter. Fallout and washout will remove nearly all of the adsorbed mercury; the vapors are prone to wide dispersal, and eventually contribute to background concentration levels. Mercury has a great affinity for organic matter, clays, and hydrous metal oxides, and in soils remains bound, provided the pH remains neutral to alkaline. Mercury may be lost from soils by volatilization; this tendency increases as the soil organic matter and moisture content decrease.

Atmospheric releases of mercury include point sources such as coal combustion, ore smelting, and solid waste incineration, and dispersive sources such as volatilization from house paints and outgassing from the earth. Mercury emissions from point sources are concentrated within 1 km of the source in surface soils and waters. This accounts for only a small percentage of total emissions, however, and the remainder is subject to dispersal according to local meteorology.

Land disposal of mercury in chlor-alkali wastes, mine tailings, coal piles, or solid wastes is a major source of mercury to the environment. However, little evidence exists to suggest that mercury enters surface or ground waters as a result of acid mine drainage, or leaching from tailings and landfills. Clays and organic matter in soils effectively reduce the quantity of mercury leaching from these systems. Soil environments favoring transportation of mercury would be low in pH and contain little clay and organic matter. Municipal landfill leachate analyses performed to date have shown mercury concentration less than or equal to 0.2 mg/l.

Mercury also reaches the soil through its use for agricultural purposes as pesticide, although this use is limited. It is lost from the soil by volatilization or retained as an adsorbed species to clays and organic matter. Phenylmercurials, which constitute most pesticidal forms of mercury, are easily leachable, as well as subject to loss by vaporization and surface runoff.

Mercury enters POTWs at an average concentration of 0.4 ug/1. Aerobic and anaerobic biological treatment partition more than 90% of the mercury into the sludge portion of the waste. Most of the remainder is adsorbed to suspended solids. Most sludges generated by POTWs are disposed of in landfills, by ocean dumping, by incineration or by land-spreading. Land-spreading of sludges to amend soils should not result in enhanced solubility or mobility of added mercury species relative to mercury already present in the soil because in both cases the mercury is strongly adsorbed, chelated or is in an insoluble form.

Discharge of mercury-containing effluents to freshwaters, whether direct or from POTWs, may result in elevated sediment concentrations for several kilometers downstream. There is a distinct prospect of methylation of the mercury in freshwater sediments. Discharges to marine waters usually result in oxidation and solubilization of the mercury followed by dilution.

3. Biological Fate

The following conclusions may be drawn concerning the fate of mercury in biota:

- Methylmercury is the most common form of mercury found in aquatic organisms.
- Methylmercury is rapidly accumulated and retained for long periods (>300 days in some species of fish).
- Both ingestion and gill absorption are exposure routes for mercury, with the former appearing to play a more significant role in upper-trophic-level organisms.
- Methylmercury tends to be associated with muscle tissue--the edible part of fish--and liver and kidneys.
- Bioconcentration levels range from one to six orders of magnitude higher than background water concentrations.
- Biomagnification of mercury appears to occur in at least certain aquatic food chains, however, further research in this area is required.

- Terrestrial plants generally do not accumulate mercury to very significant levels compared with aquatic biota. Plant residues may be higher (up to 10 mg/kg dry weight equivalent to approximately 5 mg/kg wet weight) in soils where gaseous mercury is available for uptake.

- Conversion of phenyl and other mercury compounds to methylmercury may take place in some plants.

- Clarification is needed regarding the form of mercury present in soil and its influence on uptake rates.

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CHAPTER V.

HUMAN EFFECTS AND EXPOSURE

A. HUMAN TOXICITY

1. Introduction

Mercury exists in several forms. each with different toxicity. It is important, therefore, to distinguish among the different chemical forms of mercury. Mercury compounds can be categorized as either inorganic or organic. The inorganic classification includes mercury in the form of (1) the elemental metal (Hg^0) and its vapor. (2) the mercurous ion (Hg^+) and its salts, (3) the mercuric ion (Hg^{++}) and its salts, and (4) mercuric ion complexes, which are capable of forming reversible bonds with the thiol group in proteins. The organomercurial classification includes compounds in which mercury is attached to at least one carbon atom by a covalent bond. Due to their toxicity, the most important class of organomercurials to be considered is comprised of methylmercury and related short-chain alkyl mercurial compounds.

Much information is available concerning the effects of mercury on man. The occurrence of "Minamata disease" and incidents of poisoning due to the ingestion of methyl-mercury-coated seed in Guatemala, Iraq, and Pakistan within the past 20 years have prompted additional research on the toxicity of mercury compounds. Indeed, several detailed reviews are available on the human health hazards associated with mercury exposure (Friberg and Vostal 1972, NRC 1978, Nordberg 1976, WHO 1976). Consequently, no attempt was made to present the extensive experimental data. Rather, the general findings of these reports have been summarized, supplemented by data from recently published papers in areas of concern.

2. Metabolism and Bioaccumulation

Mercury compounds may be absorbed by the body through the gastrointestinal tract, respiratory tract, or skin. The toxicity of mercury depends on the chemical form at entry in that this affects absorption, distribution, and biological half-life.

The rate of absorption, and, therefore, the oral toxicity of various compounds of mercury, increases in the following order: $Hg^0 < Hg^+ < Hg^{++} < CH_3Hg$. Metallic mercury ($Hg^0$) is not appreciably absorbed by the gastrointestinal tract (<0.01%) and the dangers of poisoning from liquid mercury by this route appear slight (Hugunin and Bradley 1975, WHO 1976). Mercuric ions (Hg^{++}) are absorbed somewhat more efficiently from the gastrointestinal tract; about 5% to 15% of the total amount ingested is absorbed by this route (Koos and Longo 1976). Mercurous ions are less water soluble than mercuric ions and, thus, are not well absorbed when ingested. The most toxic forms of mercury are the alkylmercurials, which are almost completed absorbed from the gastrointestinal tract (80-100%), but much of the amount absorbed is subsequently secreted in bile (WHO 1976, Hugunin and Bradley 1975).

Harmful levels of mercury may also be absorbed through the respiratory tract. Mercury vapor can be readily absorbed by the lungs, with about 80% of inhaled mercury taken up by the alveoli (WHO 1976). The vaporized metal quickly enters the bloodstream, where appreciable amounts persist unchanged for several minutes before undergoing final oxidation to mercuric ions. In sharp contrast to the mercuric form, methylmercury and inhaled elemental mercury vapor cross the blood-brain barrier and placenta more readily than other types of mercury (Gerstner and Huff 1977).

Cherian et al. (1978) had five human volunteers inhale 4-10.7 μCi ^{197}Hg or 1 μCi ^{203}Hg vapor in air and reported approximately 74% retention. Of the retained dose, approximately 7% was deposited in red blood cells. Another 7% of the retained dose was exhaled in expired air; the half-time for exhalation via this pathway was 14-25 hours. Cumulated urinary and fecal excretion over 7 days was 11.6% of the retained dose. Fecal excretion accounted for approximately 80% of this amount; i.e., 9.2% of the retained dose.

Monoalkylmercurials are also very volatile and diffuse readily across lung alveoli; absorption is believed to be on the order of 80% of the inhaled amount (WHO 1976). Inhaled aerosols of mercuric salts are also absorbed by the lungs, but not as readily as mercury vapor (Hugunin and Bradley 1975).

Mercury in its various forms (elemental, inorganic salts, and organic compounds) can also be absorbed through the skin, but the extent of penetration is unknown and is generally believed to be too slow a process to be of much importance in comparison with other exposure routes. Use of skin-lightening, mercury-containing facial creams by black African females, however, has resulted in episodes of hysteria, depression, uncontrollable tremor, and ataxia (Baily et al. 1977). No record of the frequency of use or amounts applied could be obtained. While this finding does indicate absorption of mercury through the skin, the possibility of some inhalation exposure cannot be excluded.

Little information is available on the distribution of mercury in human organs following well documented exposure to elemental mercury vapor, but in the brain are generally several times higher than those in liver and other organs (WHO 1976). Studies on a variety of experimental animals incicate that the kidney is the chief depository for mercury after the administration of inorganic salts or elemental mercury vapor (WHO 1976).

Urine and feces are the main routes of elimination of mercury from the body. The percentage contribution of each pathway varies according to the chemical form of mercury and the time that has elapsed since exposure. The biotransformation of elemental mercury to mercuric ion by red blood cells has been demonstrated in vitro, as has the rapid conversion of arylmercurials to inorganic mercury in the body. The short-chain alkylmercurials are converted more slowly to inorganic mercury, with methylmercury compounds being converted the most slowly (WHO 1976).

Landry et al. (1979) recently reported that diet differentially affected retention and whole-body elimination of an apparently non-toxic dose of methylmercury (0.46 mg Hg/kg, oral dose) in 8-month-old female BALB/c mice. Animals fed a chemically defined liquid diet excreted (seven days after dosing) a greater proportion of inorganic mercury to total mercury (0.91 ratio) than mice on either an evaporated whole milk diet (0.72) or standard pelleted rodent diet (0.75). Mercury concentrations in organs and blood (two weeks after exposure was initiated) generally correlated with whole-body retention of mercury.

In the case of repeated daily exposure, elimination depends on the total body burden and not on daily dose. The time needed to reach steady-state depends upon the biological half-life of the compound. In man, these values are 40 days for inorganic mercury, 58 days for elemental mercury, and 70 days for methylmercury. However, subpopulations exist with half lives as long as 120 days (WHO 1976, NRC 1978).

Mercury levels in human tissues and body fluids vary considerably, but the highest levels are generally found in the kidney and liver. Gabica et al. (1975) found that 76% of 242 tissues taken at autopsy in Idaho during 1973-74 contained detectable levels of mercury. Mean levels detected were 1.04 ug/g in kidney, 0.34 ug/g in liver, and 0.08 ug/g in brain. In general, levels of mercury were higher in women than in men once they approached or exceeded 1 ug/g tissue. Women over 65 years of age had more mercury in their tissues than had men in the same age group. These higher levels of mercury in females of advanced age remain to be explained.

In another study of 40 cadavers ranging in age from 1 year to 90 years, Schmidt and Wilber (1978) found that mercury levels in kidney tended to increase linearly with age. An earlier study of 113 people by Mottet and Body (1974), however, found no statistically significant increase in mercury burden occurring with increasing age. Regardless of the organ or age of the subject, 70% of the assays had a mercury burden of 0.25 ug/g wet tissue or less. The levels in kidney varied the most, with 29% of the concentrations above 0.75 ug/g wet tissue. Levels as high as 6.6 ug/g, 4.0 ug/g and 0.5 ug/g in kidney, liver, and brain, respectively, have been found in Japanese fetuses who succumbed to Minamata disease (Matsumoto et al. 1965).

Roels *et al*. (1978) determined the concentration of mercury in the placentas from 474 Belgian women. The median value for placenta was 1.06 ug/100g wet tissue (range of 0.11-10.31). Mercury levels in placenta were unrelated to corresponding levels in maternal and cord blood.

Yugoslavian workers engaged in the chemical industry, active mercury miners, and workers producing pesticides containing mercury accumulate significant amounts of mercury in hair. The mean values of the mercury content of hair were: 10.28 ug/g, 14.51 ug/g, and 20.94 ug/g, respectively, compared with 0.70 ug/g in the general population (Stankovic *et al*. 1977).

Thus, mercury levels in tissues of normal and exposed humans vary considerably. The mercury burden in the general population, however, appears to be below 1-2 ug/g in kidney, 0.35-0.75 ug/g in liver, and less in other tissues. Table 18 contains a summary of human tissue levels of mercury.

Nordberg (1976) summarized the known relationships between exposure levels and tissue levels, based primarily on ingestion exposures of methylmercury:

- A specific relationship exists between levels in each organ and total body burden of methylmercury.
- Definite relationships exist among the levels of methylmercury in various organs.
- Elimination is correlated with body burden, i.e., a specific fraction of body burden is eliminated per unit of time.
- A linear relationship exists between daily dose and mercury levels in blood and hair.
- Levels in blood and hair are related in a linear fashion, with the hair levels about 250 to 300 times levels in whole blood. Thus, hair is of greatest potential value as an index medium for exposure to methylmercury. Also, hair provide a record of the history of past exposure.
- At levels below which symptoms of toxicity can be observed, brain levels may be estimated accurately on the basis of blood levels.
- Urine values are of little value in estimating body burden because not only are there low levels of methylmercury present in the urine but also the relatively larger proportion of inorganic mercury present in urine introduces analytical difficulties.

TABLE 18. CONCENTRATIONS OF MERCURY IN HUMAN TISSUE

Population	Geographic Region	Tissue	No. Sampled	Distribution	Remarks	Reference
				mean ug Hg/g. wet tissue		
General population, post-mortem	Idaho 1973-1974	Kidney	94	1.04	Mercury found in 76% of tissues tested; The mean value was 0.73 ug/g with highest levels found in kidney.	Gabica *et al.* (1975)
		Liver	84	0.34		
		Brain	61	0.08		
				mean ug Hg/g, wet tissue		
General population, post-mortem, 26 wks. of gestation to 88 yrs.	State of Washington 1970-1972	Kidney	95	0.757	Irrespective of age or organ, over 70% of the assays had burdens less than 0.25 ug/g.	Mottet and Body (1974)
		Liver	95	0.250		
		Heart	57	0.102		
		Muscle	67	0.126		
		Lung	77	0.251		
		Spleen	41	0.122		
		Pancreas	32	0.065		
		Cerebellum	60	0.132		
		Spinal Cord	59	0.087		
		Skin	60	0.193		
				mean ug Hg/g. wet tissue ± S.D.		
General population, post-mortem, age 1 to 90 yrs.	Northeastern Colorado	Bone	39	0.004 ± 0.009	The amount of mercury in kidney tended to increase linearly with age with a mild increase in liver but no change in bone and muscle. Utilized atomic absorption spectroscopy.	Schmidt and Wilber (1978)
		Brain		0.000 ± 0.000		
		Kidney		1.456 ± 2.683		
		Liver		0.176 ± 0.305		
		Muscle		0.006 ± 0.009		

TABLE 18. CONCENTRATIONS OF MERCURY IN HUMAN TISSUE (Continued)

Population	Geographic Region	Tissue	No. Sampled	Distribution	Remarks	Reference
General population, Women	Belgium	Placenta	474	median 0.0106 ug/g wet tissue		Roels *et al*. (1978)
General population	Yugoslavia	Hair	---	0.70 ug/g		Stankovic *et al*. (1977)
General population, Mothers, Newborn child	Japan	Blood	9 9	22.9 ug/l ± 11.9* 30.8 ug/l ± 21.6*		Suzuki *et al*. (1971)
General population	California	Whole Blood	33	79% of all samples had concentrations below 5 ug/l; highest level reported 51 ug Hg/l.		WHO (1966)
	Ohio		40	85% of all samples had concentrations below 5 ug/l; highest level reported 240 ug Hg/l.		
	New York		87	83% of all samples had concentrations below 5 ug/l; highest level reported 45 ug Hg/l.		
General population, dieters eating tuna fish	---	Blood Hair	---	25% of population had average level of 17.3 ug/l and averaged hair concentration of 14 ug/g.		McDuffie (1975)
General population with low or zero fish consumption	---	Blood	---	<1 to 6 ug/l		Berglund *et al*. (1971)
	---	Hair	---	<1 to 5 ug/g		

*The standard deviation.

TABLE 18. CONCENTRATIONS OF MERCURY IN HUMAN TISSUE

Population	Geographic Region	Tissue	No. Sampled	Distribution	Remarks	Reference
				mean ug Hg/g. wet tissue		
General population, post-mortem	Idaho 1973-1974	Kidney	94	1.04	Mercury found in 76% of tissues tested; The mean value was 0.73 ug/g with highest levels found in kidney.	Gabica *et al.* (1975)
		Liver	84	0.34		
		Brain	61	0.08		
				mean ug Hg/g, wet tissue		
General population, post-mortem, 26 wks. of gestation to 88 yrs.	State of Washington 1970-1972	Kidney	95	0.757	Irrespective of age or organ, over 70% of the assays had burdens less than 0.25 ug/g.	Mottet and Body (1974)
		Liver	95	0.250		
		Heart	57	0.102		
		Muscle	67	0.126		
		Lung	77	0.251		
		Spleen	41	0.122		
		Pancreas	32	0.065		
		Cerebellum	60	0.132		
		Spinal Cord	59	0.087		
		Skin	60	0.193		
				mean ug Hg/g. wet tissue ± S.D.		
General population, post-mortem, age 1 to 90 yrs.	Northeastern Colorado	Bone	39	0.004 ± 0.009	The amount of mercury in kidney tended to increase linearly with age with a mild increase in liver but no change in bone and muscle. Utilized atomic absorption spectroscopy.	Schmidt and Wilber (1978)
		Brain		0.000 ± 0.000		
		Kidney		1.456 ± 2.683		
		Liver		0.176 ± 0.305		
		Muscle		0.006 ± 0.009		

TABLE 18. CONCENTRATIONS OF MERCURY IN HUMAN TISSUE (Continued)

Population	Geographic Region	Tissue	No. Sampled	Distribution	Remarks	Reference
General population	New York	Urine	363	80% samples below 0.5 ug/1; highest level reported 97 ug/1		WHO (1966)
	California		31	87% samples below 0.5 ug/1; highest level reported 15 ug/1		
	Ohio		40	93% samples below 0.5 ug/1; highest level reported 221 ug/1		

3. Animal Studies

a. Carcinogenicity

Little information is available on the carcinogenicity of mercury compounds. Schroeder and Mitchener (1975) reported no significant difference in tumor frequencies between control, unexposed, and exposed random-bred albino Swiss mice following lifetime exposures to methylmercury in drinking water (5000 ng/ml for 70 days, then 1000 ng/ml thereafter).

Prolonged exposure of mice to 1000 ng/g or 10,000 ng/g methlymercury in their feed also did not alter the course of neoplasia following inoculation with Rauscher leukemia virus (Koller 1975).

Localized sarcomas were reported in rats injected intraperitoneally with metallic mercury (Druckrey et al. 1957), but no metastases were observed. Carcinogenesis resulting from injection in which tumors are induced only at the site of application is generally regarded as irrelevant to human exposure.

The available data (a single lifetime exposure in one species, the mouse) are inadequate to permit a reliable assessment of the carcinogenic potential or mercury, but at this time mercury is not indicated as a carcinogen.

b. Mutagenicity

Mutagenicity studies conducted in plants and laboratory animals have shown the ability of methylmercury to block mitosis in plant cells, human lymphocytes treated *in vivo*, and human cells in tissue culture and to cause chromosome breakage in plant cells and point mutations in *Drosophila* (NAS 1978, U.S. EPA 1979, Friberg and Vostal 1972, Voss et al. 1978, Mathew and Al-Doori 1976).

Reports of mutagenic effects in humans resulting from methylmercury exposure are few. Skerfving et al. (1974) reported a statistically significant correlation between the frequency of chromosome breaks and blood mercury concentrations in individuals with elevated blood methylmercury levels (range 13-1100 ng/g) due to the ingestion of fish containing methylmercury.

Recently, Popescu et al. (1979) reported that the incidence of chromosome aberrations (mostly acentric fragments) in peripheral blood of 22 men exposed to either mercury vapor or organic mercury was significantly higher than in controls. Although the number of chromatid gaps and breaks was increased in the exposed men (38 versus 16 in controls), the increase was not statistically significant. Mercury concentration in the chemical plant in which these men were exposed had ranged between 0.15 mg/m^3 and 0.44 mg/m^3 during the past year.

Rozynkowa and Raczkiewicz (1977) found severe mitotic toxicity in human lymphocytes exposed to 40 ug/ml methylmercuric chloride for 2 hours in culture. This type of damage is probably not of mutagenic significance, since the cells cannot survive and carry the alteration of the genetic material.

Similarly, Umeda and Nishimura (1979) found that mercuric chloride was relatively toxic to FM3A mammary mouse carcinoma cells, but failed to induce chromosomal aberrations at subtoxic concentrations (3.2×10^{-5}M).

In another study, Fiskesjo (1979) tested the mutagenicity of two organic mercury compounds, methylmercuric chloride (MMC) and methoxyethyl mercury chloride (MO) in the Chinese hamster cell line, V79-4. A weak mutagenic effect was noted, but acute toxic effects obtained with both compounds limited dose-response curves for mutagenicity to a very narrow concentration range:

MMC 0.1 mg/kg (no effect) to > 0.5 mg/kg (poor survival);
MO 0.05 mg/kg (no effect) to > 0.3 mg/kg (poor survival).

At 0.2 mg/kg, the number of mutants per 10^4 survivors was 18.3, 30.7, and 4.3 for MMC, MO and controls, respectively.

Casto *et al*. (1979) tested mercuric chloride for its capacity to enhance transformation of Syrian hamster embryo cells by a simian adeno virus, SA7. Mercury showed moderate enhancement of viral transformation following 18-hour exposure to 0.05 mM; an enhancement ratio of 5.6 above control was recorded at 0.05 mM.

In summary, methylmercury has been shown to be a weak mutagen in *Drosophila*. It can interfere with mitotic and meiotic chromosome segragation in plants and animals and has been reported to produce chromosomal aberrations *in vitro* in lymphocytes of individuals exposed to methylmercury. Contradictory data exist on its ability to induce chromosomal breaks in man. The significance of these observations for human health remains unclear.

c. Adverse Reproductive Effects

Due to its great affinity for sulfhydryl groups, mercury poses a particular hazard to the developing embryo. Methylmercury readily crosses the placental barrier, inducing a variety of developmental anomalies and death. The mechanisms by which methylmercury interferes with fetal development, growth, and viability are not well known. Although prenatal exposure to mercury has proved to cause a number of harmful effects on the human fetus, to date, epidemiological studies from human poisoning episodes have been inadequate to fully define dose-response relationships or to conclude that the full range of possible teratogenic effects has been identified.

Embryotoxicity and teratogenicity of mercury in animals, however, have been well documented (Mottet 1978, Koos and Longo 1976). Hamsters, rats, or mice given acute high doses of organic mercurials during sensitive periods of gestation have demonstrated a spectrum of malformations, including cleft lip and/or palate, micrognathia, encephalocele/excencephaly, microphthalmia, rib fusions, and syndactyly. Growth retardation, litter resorption, and stillbirths occurred frequently. Methylmercury compounds particularly affect nervous tissue, resulting in cerebellar malformations, nerve degeneration, and hydrocephalus. Differences in species, dosing regimen, and chemical form of mercury administered, however, make direct comparisons among studies difficult.

Recent studies serve as typical examples of the effect of mercury on the developing embryo. Fuyuta et al. (1978) administered daily oral doses of 2.5 mg/kg, 5.0 mg/kg, 6.0 mg/kg, or 7.5 mg/kg methylmercuric chloride (MMC) to pregnant C57BL mice on days 6-13 of gestation and found that it was teratogenic at the lowest dose tested. The highest dose, 7.5 mg/kg MMC, was embryocidal (i.e., 98.7% dead and resorbed embryos). At a dose of 6 mg/kg, a high incidence of fetal death (34.2%) was noted, while both the 6 mg/kg and 5 mg/kg groups showed decreases in fetal body weight and marked increases in malformations (cleft palate, fused thoracic vertebrae). The incidence of malformations for 6 mg/kg, 5 mg/kg, 2.5 mg/kg and 0 mg/kg MMC groups was 97.9, 75.7, 11.3 and 0%, respectively.

A concurrent experiment conducted with Wistar rats by these investigators resulted in a high incidence of fetal deaths and resorptions (42.4%) and an 80.3% incidence of malformations, especially cleft palate, generalized edema, and brain lesions, in rats given 7.5 mg/kg orally on days 7-14 of gestation. Rats similarly treated with 5 mg/kg, 2.5 mg/kg, or 0 mg/kg MMC had incidences of malformations of 6.8, 0.0 and 0.4%, respectively.

Olson and Boush (1975) reported decreased learning capacity in Holtzman rats exposed pre- and post-natally to 2 mg mercury/kg of diet.

Olson and Massaro (1977) reported that methylmercury (5 mg Hg/kg maternal body weight) given subcutaneously to gravid Swiss Webster CFW mice on day 12, hour 6 of gestation induced a high incidence of cleft palate in fetuses examined on days 15 (72%), 16 (62%), and 17 (40%). Palate closure (100%) occurred by 14 days in control animals.

Eccles and Annau (1978) orally exposed Long Evans rats to 0 mg/kg, 5 mg/kg, or 8 mg/kg methylmercury in utero on day 7 of gestation. At a dose of 8 mg/kg, 40% of litters were resorbed, but litters that were delivered were of normal size and weight.

Gale (1979) injected LVG hamsters subcutaneously with a single 15-mg/kg dose of mercuric acetate at 8 A.M., Noon or 5 P.M. on day 7, 8, or 9 of gestation. Treatment resulted in fetal death and external, internal, and skeletal abnormalities in survivors. Treatment at each of the nine injection times was equally as effective in producing many, but not all defects. Pericardial cavity distension and ventral body wall defects were observed in fetuses taken on day 12, but not in those gathered on day 15, a finding suggesting a transient nature to this defect. Fetuses taken on day 15 exhibited cleft palates, hydrocephalus, skeletal defects, and abnormal hearts characterized by dilation of the walls of the right ventricle and/or conus cordis.

Mottet (1978) examined the effects of chronic low subcutaneous doses of methylmercuric hydroxide to the developing rat at dose levels ranging from slightly in excess of the environmental burden (2 mg/kg maternal body weight) to overt clinical toxicity (16 mg/kg maternal body weight). Rats were dosed from day 0 to day 20 of gestation. No detectable increase in specific malformations was noted, but a dose-related decrease in fetal size was observed; i.e., 4.2 g, 3.7 g, and 2.1 g for the control, 2 mg, and 16 mg Hg/kg levels. Decreased size appears to be associated with a decreased number of cells per organ or tissue. Fetal death and fetal mercury burden were also dose-related.

Decreased fertility has also been noted in male mice given a single intraperitoneal dose of 1 mg/kg methylmercury hydroxide. Fertility profiles from serial matings suggest an effect on spermatogonial cells and premeiotic spermatocytes (Lee and Dixon 1975). Similar results have been reported by Suter (1975) and Ramel (1972) at somewhat higher doses.

In summation, elemental and methylmercury have been shown to readily cross the placenta, inducing a variety of developmental anomalies and fetal death. Laboratory animals exposed to organic mercury _in utero_ exhibit a wide spectrum of malformations including cleft palate, micrognathia, encephalocele, etc., at doses as low as 2.5 mg/kg maternal body weight. Methylmercury compounds appear to be particularly predisposed to concentrate in nervous tissue, producing cerebellar malformations, nerve degeneration and hydrocephalus.

d. Other Toxicological Effects

The toxicologic responses noted after the administration of mercury vary depending upon the formulation or the chemical form administered (organic or inorganic). Regardless of the form of the chemical, however, the two major responses noted after mercury administration are neurotoxicity and renal damage.

The neurotoxic effects of mercury-containing compounds are well characterized for only a few of the more common forms, e.g., methylmercury and inorganic mercurials such as $HgCl_2$. Considerable variation among mercury compounds in gastro-intestinal absorption, metabolism, and elimination from the body, as well as differences in uptake, distribution, and elimination from the brain and other nervous tissue, all serve to produce different neurological responses.

The neurotoxic effects follow from the ability of mercury compounds, both organic and inorganic, to penetrate, bind, and significantly alter biological membranes. Damage to the blood-brain barrier, a highly selective complex of biological membranes, reduces the active transport of crucial nutrients such as amino acids, and permits the penetration of blood solutes normally barred from the cerebro-spinal space, and these cause neurocellular disintegration (Chang 1977). The monovalent alkyl mercuric ion ($R-Hg^{+}$) and the divalent mercuric ion (Hg^{++}) have a strong affinity for sulfhydryl groups of proteins, and the cellular membranes are rich in sulfhydryl groups. Electronmicroscopic studies have shown large amounts of mercury localized to membranes of the mitochondria, golgi apparatus, endoplasmic reticulum, and nuclear envelope (Chang et al. 1972).

Studies on experimental animals have provided information on mechanisms and sites of action for the mercurials, but need to be interpreted carefully with respect to dose-effect relationships. There is considerable species variation in the uptake of mercurials into the brain from the blood. At approximately steady state, the blood-brain concentration ratio is approximately 10-15 for rats, 1 for mice, 1-2 for cats, 0.5 for dogs and pigs, and 0.1 for monkeys (Chang 1977). Neurotoxic signs occur in most species at brain concentrations within an order of magnitude of each other (i.e., between 1-10 mg/kg) although corresponding blood concentrations may differ widely due to species differences in blood to brain ratios (U.S. EPA 1979).

There is evidence, however, that primates may be more sensitive to low levels of mercury in the brain than rodents. In rats, motor deficiency has been detected at brain levels no lower than 5-10 mg/kg following divided doses of methylmercury totalling 34 mg/kg of body weight. In young monkeys dosed with divided or single doses of methylmercury (4.6-6.9 mg/kg), severe neurotoxic effects were observed when brain levels had reached only 1-2 mg/kg. These monkeys became physically incapacitated and comatose at brain levels of 6-12 mg/kg (Hoskins and Hupp 1978). Species differences in biological half-lives of mercury compounds, together with the differences in blood-brain barrier and possible intrinsic neural sensitivity, all contribute to species variation in dose-effect relationships for neurotoxic symptoms.

The other major irreversible effect associated with mercury exposure is renal damage. Irrespective of chemical form at entry, kidneys concentrate more mercury than any other organ, often to an extent that is incompatible with normal renal function and morphology. Renal damage can result in oliguria, anuria, uremia, and death. Morphologic damage to renal tubule cells has been demonstrated in rats following either acute or chronic exposure to methylmercury (Fowler and Woods 1977, Hinglais et al. 1979, Fowler 1972). Similar results have been reported following treatment with other mercurials (Friberg and Vostal 1972).

A recent report by Goldman and Blackburn (1979) indicates that mercury may also influence thyroid function in the rat. Oral administration of 3 mg/day of mercuric chloride for 6 consecutive days accelerated the release rate of thyroidal radioiodine (^{131}I). Administration of approximately the same dose (2.5 mg/day) by stomach tube for 40 days resulted in continued enhancement of thyroid activity. A reduction in the fraction of labelled triiodothyronine (T_3) was found and may indicate a coupling defect in the synthesis of T_3 exerted by mercury. Subchronic exposure to 100 mg/kg mercuric chloride in the diet for 90 days (which approximated the 2.5-mg/day dose by gavage), however, resulted in manifest signs of mercury poisoning, together with decreased thyroid radioiodine uptake and depression of thyroid secretion rate, which was irreversible even after 3 months on a control diet.

Thus, the toxic effects of mercury have been shown to vary depending on chemical form administered, species variation in absorption, brain uptake, etc. Neurotoxicity and renal damage are the two major toxic effects noted after mercury exposure. Neurotoxic signs occur in most species at brain concentrations between 1 and 10 mg/kg with primates apparently more sensitive to the minimally effective brain concentration than rodents, morphological damage to renal tubule cells have been demonstrated in laboratory animals following either acute or chronic mercury exposure irrespective of chemical form at entry.

e. Interactions With Other Metals

A complete discussion of the complex interactions of mercury with other metals is beyond the scope of this report. There is no question, however, that the toxic effects of mercury are modified to some extent in the presence of selenium and other metals.

Selenium appears to diminish the acute and subchronic toxicity of mercury in rodents (Skerfving 1978). Excess mercury provokes a pattern of selenium retention similar to that found in cases of selenium deficiency (Kristensen and Hansen 1979). Dietary selenium also influences tissue distribution of inhaled mercury vapor in rats (Nygaard and Hansen 1978). The protective mechanism of selenium against mercury toxicity is not well understood, but selenium appears to eliminate the stimulation of metallothionein biosynthesis induced by mercury (Chmielnicka and Brzezhicka 1978). Of note, however, is the finding that selenium-treated animals remain unaffected even when they have attained tissue mercury levels otherwise associated with toxic effects. This subject has been reviewed in detail by Skerfving (1978), Berlin (1978), and Parizek (1978).

With respect to other metals, manganese, which is present in all waters, has been shown to be an avid scavenger of mercury, and iron has a similar action (Anderson 1973, Lockwood and Chen 1973). Conversely, the toxic effects of mercury are accentuated by the presence of copper (Corner and Sparrow 1956).

4. Human Studies

Both natural and cultural sources contribute to widespread, low-level mercury contamination of the environment, as discussed in Chapter III. As a result, all humans are exposed to low levels of mercury through inhalation and most are also exposed to low levels through ingestion of water and food. Occupational exposures and effects have been recognized, if not well characterized, for centuries in some cases. These are discussed at more length in Section b. Chronic Exposure, as are three incidents of widespread human toxicity due to ingestion of methylmercury.

A number of other accidental or incidental cultural sources have been reported. These include poisonings due to inhalation of elemental mercury vapor from broken thermometers (Agner and Jans 1978), from mercurochrome therapy for an infected umbilicus (Yeh et al. 1978), due to release of mercury from amalgam dental fillings (Gay et al. 1979), and due to ingestion of small mercury batteries by children (Reilly 1979, Barros-D'Sa and Barros-D'Sa 1979). While a considerable number of such incidents have been reported, most of the reports have been anecdotal in nature, often not including information on the dose received (although reasonable estimates may be made later in some cases) and rarely provide information on incidence or exposed population, although again estimates of differing reliability may be made later.

a. Acute Exposure

The acute symptoms resulting from the ingestion of any mercury-containing compound may initially be noted by an ashen-grey appearance of the mouth and pharynx. This condition results from precipitation of the protoplasm of the mucous membrane, and is often accompanied by a burning sensation in the mouth and throat and eschar formation on the mouth and lips. Extreme salivation and thirst often follow. The mucous membrane of the stomach is similarly affected. Consequently, gastric pain, nausea and vomiting of blood-stained mucus result. If a high concentration of mercury reaches the small intestine, severe, profuse, and bloody diarrhea result, often accompanied by shreds of intestinal mucosa. Due to loss of fluids and electrolytes, shock may be accompanied by a rapid, weak pulse; cardiac arrhythmias; cold, clammy skin; pallor; slow breathing; and peripheral vascular collapse (D'Itri 1972).

If the patient survives, the following delayed actions may occur within 1 to 14 days: ulcerative colitis; salivary gland swelling; excessive salivation; metallic taste, stomatitis, foul breath, loose teeth; soft spongy gums; and a blue-black gum line caused by a mercury-sulfhydryl complex. Systemic signs, referable to the central nervous system, include lethargy, excitement, hyper-reflexia and tremor (Harvey 1970, D'Itri 1972). Oliguria is often present, with anuria, uremia, albuminuria, hematuria, proteinuria, and acidosis. Death at this stage is ascribed to uremia. Autopsies reveal inflammation and extensive corrosion along the alimentary tract, severe renal tubular necrosis, and possibly, central necrosis of the liver (D'Itri 1972).

b. Chronic Exposure

The onset of chronic mercury poisoning is often slow and insidious, typically beginning with progressive numbness of the distal parts of the extremities and often of the lips and tongue. This is followed by an ataxic gait, clumsiness of the hands, dysarthria, dysphagia, deafness, and blurring of vision. Voluntary movements are limited in most individuals although muscle atrophy is rare. Spasticity and rigidity are often present, muscle stretch reflexes are usually preserved or become hyperactive, and extensor plantar responses are occasionally elicited during the later stages. Insomnia, agitation, hypomania, and the loss of emotional control are frequently noted and most individuals have abnormal involuntary movements, including choreoathetosis, myoclonus, and coarse resting and action tremors (D'Itri 1972).

Three major outbreaks of methylmercury poisoning have occurred in man. In Minamata, Japan, the poisoning was caused by marine fish. In Niigata, Japan, the methylmercury was carried by freshwater fish, and in Iraq, methyl-mercury-contaminated grain was ingested by the rural population. Individuals involved in these outbreaks have demonstrated a wide range of neurologic symptoms. Feelings of malaise have been observed, progressing to severe bodily discomfort with muscular weakness, paresthesia, loss of coordination of the digits, ataxia, speech disturbances, disturbances of vision (blurring and constriction of field of vision) and loss of hearing, among many other manifestations of neurotoxicity. Character disorders and mental deficiency have also occurred. Symptoms were similar in children and adults. Recovery from methylmercury intoxication is inversely related to the severity of symptoms, and ranges from complete functional recovery in persons experiencing minor symptoms, such as slight paresthesia, to indefinitely protracted physical and mental disabilities in severely poisoned individuals. Tokuomi (1968) reported that neurological abnormalities were still apparent after 10 years in some patients who had experienced Minamata disease. Tremor of fingers was apparent in 70% of the patients. Takeuchi et al. (1970) noticed that some symptoms of central and peripheral nervous system disturbances persisted unchanged, while symptoms such as mental abnormalities worsened over 10 years in patients with Minamata disease.

Evaluation of human populations following large-scale exposure to methylmercury compounds has been used to estimate the threshold dose and corresponding blood levels that produce certain neurologic effects. These estimates have been used to estimate the "safe" exposure levels at which neurological symptoms should not occur. The Swedish Expert Group (Berglund et al. 1971) made two estimates of the critical daily intake based on the Japanese exposures at Minamata Bay and Niigata. Using the metabolic method, which incorporated data on brain levels, absorption, distribution, and a biological elimination half-life of 70 days, they obtained a critical daily intake of ~ 10 ug/kg. The epidemiologic method, which correlated blood levels and clinical symptoms in both poisoned and non-poisoned individuals with methylmercury consumption in fish, gave an estimate ~ 5 ug/kg. When the lower of the two estimates is used with a safety factor of 10, 30 ug/day of methylmercury appears to be a safe level of intake for a 70-kilogram man. This would correspond to an acceptable level of methylmercury in blood of approximately 20 ng/ml. Subsequent epidemiologic studies summarized in Table 19 seemed to verify that the 200 ng/ml blood level is approximately the level at which certain neurologic effects would begin to occur. It is not known whether a safety factor of 10 is sufficient, particularly for developing fetuses and infants (see Chapter V.4.c.) and in some individuals in whom biological half-lives for mercury compounds are as long as 120 days (Al-Shahristani and Shibab 1974).

TABLE 19. CLINICAL CORRELATIONS OF NEUROTOXICITY AND LEVELS OF MERCURY IN BLOOD

Blood Levels (ng/ml)	Incidence of Neurological Symptoms	Reference
200[1]	~ 5%	Bakir *et al*. (1973)
5.330	42%	Clarkson (1975) Harada *et al*. (1976)
>50[2]	<50%	Barbeau *et al*. (1976)
11-275 mean 82	0%	Turner *et al*. (1974)

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[1] Extrapolated background level for paresthesia (earliest clinical sign)

[2] Considered suspect because of likely inclusion of patients suffering from alcoholism.

WHO (1976) has established a provisional weekly intake of 0.3 mg total mercury, of which no more than 0.2 mg should be present as methylmercury. These doses correspond to daily intakes of 43 ug and 29 ug, respectively for a 70 kg person, slightly higher than those recommended by Berglund *et al*. (1971).

Chronic inhalation exposure to mercury vapor in the workplace has resulted in tremors, mental disturbances and gingivitis at air concentrations above 0.1 mg/m^3; lower concentrations (0.06 to 0.1 mg/m^3) are associated with such non-specific signs as insomnia, loss of appetite, weight loss. Occupational exposure to an air concentration of 0.05 mg/m^3 mercury vapor would be equivalent to continuous environmental exposure to an ambient air level of ~ 0.015 mg/m^3 (based on a daily ventilation of 10 m^3 during working hours, 20 m^3 for a 24-hour day, 225 working days/year) (WHO 1976).

c. Adverse Reproductive Effects

The human fetus appears to be very susceptible to mercury poisoning. The information available concerning the human reproductive-teratogenic effects of mercury is epidemiological in nature; the Minamata and Niigata studies are prime examples. Although these studies indicate that organic mercury passes the human placenta, the actual concentration of mercury ingested by the mother, together with the duration of exposure, cannot be determined.

Six percent of the children born near Minamata Bay between 1954 and 1959 were afflicted with mild to moderate spasticity, ataxia, chorea, coarse tremors, seizures and severe intellectual deficiencies (Scanlon 1972). Since mercury can be excreted in breast milk (Berlin and Ullberg 1963), many of these affected children may have acquired high mercury levels both *in utero* and from their mothers' milk.

In all 19 reported cases of congenital infantile cerebral paresis in Minamata and Niigata, the mothers displayed few or no clinical neurological symptoms (Eyl *et al*. 1970). This absence of symptoms may be due partially to the fact that fetal erythrocytes concentrate higher levels of mercury than do maternal erythrocytes.

Typically, abnormalities were recognized at the beginning of the sixth month after birth. Symptoms included instability of the neck, convulsions, and failure of the eyes to follow. Patients also developed severe mental and neurologic symptoms including: intelligence disturbance (100%), disturbance of body growth and nutrition (100%), hyperkinesia (95%), hypersalivation (95%), paroxysmal symptoms (82%), strabismus (77%) and pyramidal symptoms (75%). Clinical evidence of fetal brain damage was observed when maternal blood levels of mercury of approximately 400 ng/ml were achieved (Harada 1978, NRC 1978).

In 1971, barley and wheat grain treated with methylmercury were used to make bread containing about 4 mg of Hg/loaf; ingestion of this bread resulted in a widespread epidemic of mercury poisoning in Iraq. Infants born to women pregnant during this period suffered severe brain damage. Breast milk was shown to contain 5-6% of maternal blood Hg levels and may have contributed to the problem (Koos and Longo 1976).

The harmful fetal effects of methylmercury were further implicated in a male child born to a woman who had ingested contaminated pork during the third through sixth months of pregnancy. The meat became contaminated after hogs were inadvertently fed seed grain treated with a methylmercury fungicide. Examinations of the mother were "normal" for the remainder of the pregnancy, except for elevated levels of mercury in the urine (0.18 mg/l at 8 months). The male infant (3.06 kg) was delivered at term. Intermittent gross tremulous movements of the extremities developed within 1 minute of birth and persisted for several days. The child was normal in all other respects except for a high urinary level of mercury (2.7 mg/l at 1 day of age). At 6 weeks, the infant was hypertonic and irritable; no mercury could be detected in his urine. At 8 months of age, the baby was irritable, began to have myoclonic seizures, and was now hypotonic, grossly retarded, and had nystagmoid eye movements without evidence of visual fixation. Since this infant was never breast fed, this case presumably resulted from actual intrauterine poisoning with organic mercury. The mother was asymptomatic, in striking contrast to the symptomatology seen in the infant, a finding that may indicate a special susceptibility of the developing human nervous system to damage from mercury (Snyder 1971).

The most perplexing aspect of this circumstantial evidence that methylmercury is a teratogenic agent for human fetuses is the lack of symptomology in the mother during pregnancy while the child has marked neurotoxic symptoms soon after birth. Whether this divergent response to methylmercury exposure is due to the ability of the fetus to concentrate mercury *in utero*, or because the developing nervous system of the fetus may be hypersensitive to the toxic effects of methylmercury, cannot be determined. Both factors may play a role in methylmercury's teratogenicity since the embryo has been shown to concentrate greater amounts of mercury in red blood cells than its mother; the nervous system may be more sensitive because myelination may not have been completed at the time of exposure.

5. Overview

Mercury compounds may be absorbed through the gastrointestinal tract, respiratory tract and through the skin. In man, toxicity increases in accordance with the extent of absorption, i.e., with increasing toxicity of mercurial compounds as follows: $Hg^0 < Hg^+ < Hg^{++} < CH_3Hg$. Methylmercury is of special biological significance because, unlike its inorganic counterparts, it is readily absorbed from the gastrointestinal tract (80-100%), gains ready access to the placenta, crosses the blood-brain barrier, and is eliminated from the human body much more slowly than other mercurials. Biological half-lives in man are 40 days for inorganic mercury, 58 days for elemental mercury, and 70 days for methylmercury. Mercury levels in human tissues and body fluids vary considerably, but the highest levels are generally found in kidney and liver.

Except for the production of local sarcomas at the point of injection of metallic mercury in rats (findings that are generally regarded as irrelevant to human exposure), there are no data available to indicate that mercury compounds are carcinogenic.

Methylmercury is a weak mutagen in *Drosophila* and can interfere with mitotic and meiotic chromosome segregation in plants and animals. Methylmercury also produces chromosomal aberrations in human lymphocytes *in vitro* and has been implicated in the induction of chromosomal breaks in man.

Mercury poses a particular hazard to the developing embryo. Elemental and methylmercury readily cross the placental barrier, inducing a variety of developmental anomalies and fetal death. A wide spectrum of malformations including cleft palate, micrognathia, encephalocele, etc., have been produced in laboratory animals exposed to mercury *in utero* at doses as low as 2.5 mg/kg maternal body weight. The human fetus, and in particular, the fetal nervous system, appears to be particularly susceptible to methylmercury as indicated by the Minamata and Niigata episodes.

Most of the human data available on mercury exposure are epidemiological in nature. The critical organ systems in man are the central nervous system and the kidneys. The onset of chronic poisoning is often slow and insidious and typically begins with numbness of the distal parts of the extremities, and often of the lips and tongue. This is followed by progressive neurological disturbances including dysarthria, ataxia, concentric constriction of the visual fields, blurred vision, blindness, deafness, and ultimately, death.

A critical daily intake of 30 ug Hg, which corresponds to a mercury blood level of 20 ng/g, has been estimated to be a safe intake for an average 70-kg man. However, there is some disagreement as to a "safe intake." A blood mercury concentration of 200 ng/g is the approximate blood level at which observable neurological effects occur.

B. EXPOSURE

1. Introduction

The previous section describes the toxicity of mercury to humans, especially methylmercury. Because effects have been observed following exposure to low levels of mercury, there has been a great emphasis on developing exposure estimates over the past 5-10 years. Since numerous authors have taken considerable effort to review and analyze available data, their work is primarily summarized here, without going into great detail. For more information and background, the reader is referred to NRC (1978), U.S. EPA (1979), and WHO (1976) for excellent reviews of this area.

Though much still remains to be learned regarding the toxicity of mercury, it is clear that effective exposure and toxicity depend on the route of exposure and the chemical form of mercury. Thus, exposure estimates for different routes cannot simply be summed. Therefore, the following section considers separately each exposure route and the form of mercury generally associated with it.

2. Ingestion

a. Drinking Water

The intake of mercury in drinking water is generally considered to be low. Battelle (1977) cites an EPA survey of finished drinking water conducted in 1975-76. Of the 512 water supplies sampled, 460 were less than the detection limit of 0.5 ug/l. Six samples had concentrations greater than 2 ug/l. Thus, according to these data, most persons consuming 2l per day would be exposed to less than 1 ug/day in drinking water, and a very small subpopulation would receive 4 ug/day.

The primary form of mercury in drinking water is probably soluble inorganic compounds. NRC (1978) reviewed available data for evidence of methylmercury in natural waters. The authors found very low levels, generally less than 0.0002-0.001 ug/l in nonpulluted waters (See Chapter IV).

b. Food

Food has been considered to be the primary route of human exposure to mercury. Almost all of the methylmercury in the human diet comes from fish; however, other foods may contribute to the total mercury exposure (NRC 1978). The U.S. FDA has recently raised the action level* for mercury in fish, shellfish, crustaceans, and other aquatic animals from 0.5 to 1.0 mg/kg (FR 44:4012). NRC (1978) reports that this level is generally exceeded only by the larger marine species and freshwater species from particularly contaminated areas. For example, fishing locations and catches are restricted in many areas of the U.S. Figure 14 shows the status of state restrictions on fishing due to mercury in 1977. Appendix B contains an update of the status of these fisheries. At this time many of them remain restricted, though restrictions have been lifted in some states.

In addition, numerous incidents of food contamination due to mercury have been reported. State agencies reported 19 such incidents from 1968 to 1978, and federal agencies reported 85 cases in the same time period (OTA 1979). These incidents are defined as cases in which an agency has taken regulatory action against a contaminated food, and, therefore, represent only some portion of the total incidents that have actually occurred, most of which are probably attributable to contaminated fish that had exceeded the previous action level of 0.5 mg/kg set by FDA in 1969.

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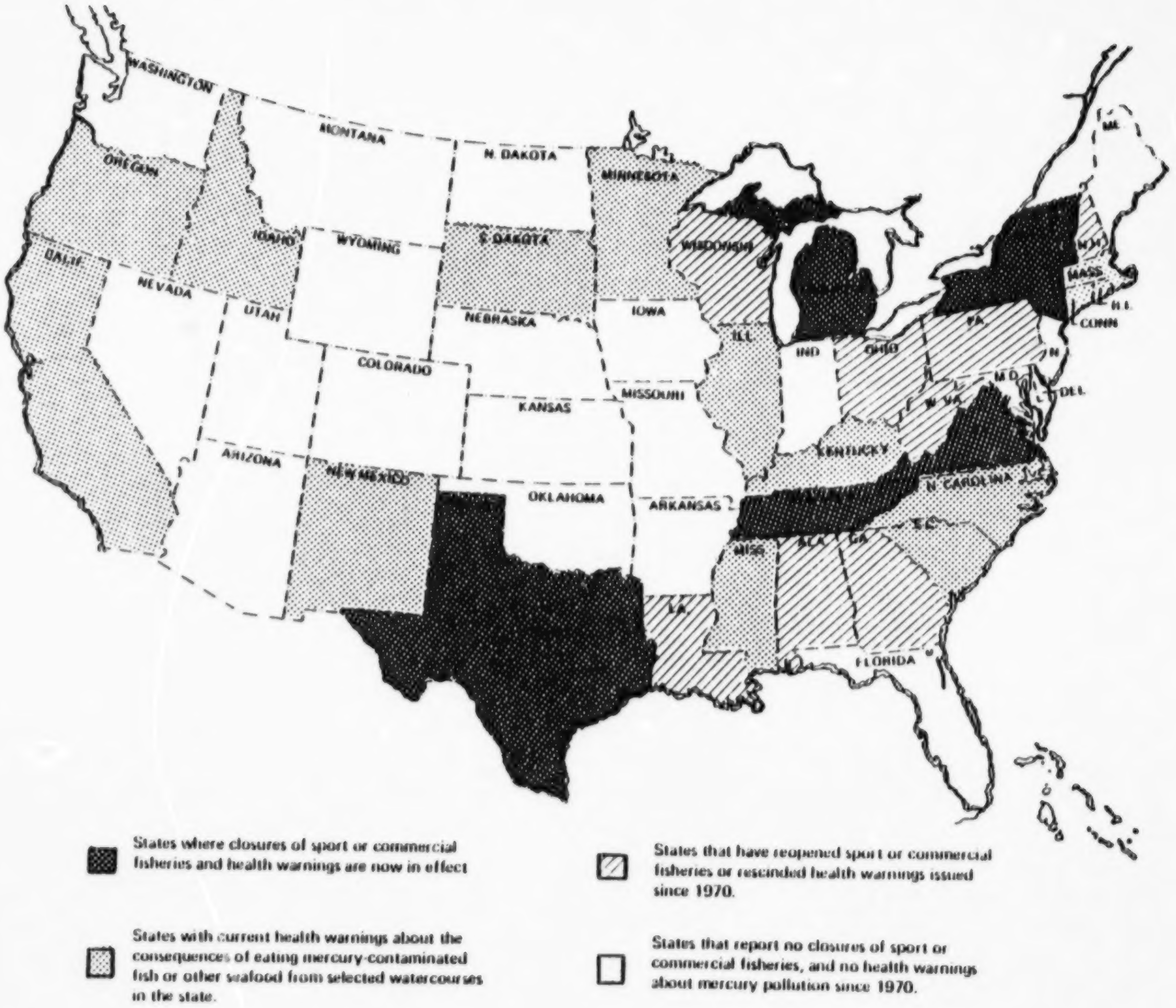
*Level at which FDA can take action to remove fish from the market place.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NEBRASKA
KANSAS
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DEL.
N.J.
N.H.
MASS.
R.I.
CONN.
ME.
▩ States where closures of sport or commercial fisheries and health warnings are now in effect
▧ States that have reopened sport or commercial fisheries or rescinded health warnings issued since 1970.
▒ States with current health warnings about the consequences of eating mercury-contaminated fish or other seafood from selected watercourses in the state.
☐ States that report no closures of sport or commercial fisheries, and no health warnings about mercury pollution since 1970.

Source: NRC (1978)

FIGURE 14 STATUS OF FISHERY RESTRICTIONS AND CLOSURES IN THE UNITED STATES, 1977

Though there are considerable data concerning mercury contamination of fish (see Chapter IV, WHO 1976, and NRC 1978), most of it is 5 or more years old. Levels do not appear to vary much in marine species with time, but levels in freshwater species vary to a greater degree. The most recent and comprehensive survey of levels of mercury in fish was conducted by Hall et al. (1978). These authors found that muscles of most finfish had mean mercury levels below 0.3 mg/kg. Thirty-one species contained mean levels in excess of the previous action level (0.5 mg/kg); however, less than 2% of the U.S. catch was in excess of the 0.5 mg/kg action level. Detailed information on contamination levels can be found in Hall et al. (1978).

These data on concentrations of mercury in aquatic organisms, as well as data from other sources, have been used by other authors to calculate exposures to mercury for various subpopulations. The various assumptions used in each resulted in widely variable estimates. The most detailed estimate was performed by NMFS (1978). They used the extensive monitoring of Hall et al. (1978), as well as a fish consumption survey which included 25,947 participants. The amounts of each species consumed by an individual were combined with the concentration of mercury for that species as measured in the Hall et al. (1978) survey (using the value at the 95% confidence limit) in order to estimate the intake in an individual. NMFS (1978) used various action limits to determine the chance of a person's exceeding their Allowable Daily Intake (ADI). The ADI was 30 ug/70 kg, adjusted to individual body weights. The results of this exercise are shown in Table 20 These data suggest that if the sample of the human population, as well as the seafood survey, are representative, approximately 0.1-0.2% of the population is exposed to mercury in seafood (with 95% confidence limits) amounting to more than 0.43 ug/kg body weight/day.

Other authors (U.S. EPA 1979, NRC 1978) have used levels in tuna for calculating mercury intakes, using the assumption that tuna makes up 75% of the fish intake. This would appear to underestimate exposure for consumers of large amounts of seafood. The consumption patterns of the persons exceeding their ADI (NMFS 1978) showed that, in many cases, the greatest proportion of mercury intake was due to consumption of less common, but more highly contaminated species of fish. Table 21 shows intake for two such persons, who represent the maximum calculated intakes based on consumption patterns in the survey. The upper limit daily intake in the table was calculated using the mercury concentration value at the 95% confidence level for a given species, while the maximum intake calculation in the table uses the maximum reported contamination for a given species and assumes that levels in fish consumed are not being restricted (no action limit). This table shows that a small percentage of the population (<.008% may be receiving intakes of mercury in seafood in excess of 100 ug/day. With the 0.5 ug/g action limit, the upper limit daily intake was reduced to 80 ug/day, but the maximum (again with the action limit) for the same individual was 222 ug/day.

TABLE 20. PERCENT OF POPULATION EXCEEDING THE RECOMMENDED ADI FOR MERCURY DUE TO FISH CONSUMPTION[1]

Group	No. Persons	No Action Limit %	0.5 mg/kg action limit %
Total	24,652	0.19	0.11
Women of child-bearing age	3,884	0.15	0.10
Children	4,423	0.34	0.20

---

[1] Data based on intakes caluclated using consumption data for population combined with 95% CL for concentration data for species consumed. A 75% compliance with the action limit was assumed.

Source: NMFS (1978).

TABLE 21. MAXIMUM INTAKE OF MERCURY FOR TWO FISHEATERS

Person	Species	g/serving	Serving/ month	Mercury Concentration in Fish (ug/g)		Upper Limit Daily Intake (ug) 95% confidence limits		Maximum Intake	
				Avg.	Max.	No action limit	0.5 ug/g action limit	No action limit	0.5 ug/ action limit
Person 1	Pike	206	15	0.01	1.7	78.84	51.37	217	141
	Bass	167	3	0.75	2.0				
	Perch (marine)	144	2	0.13	0.59				
	Not identified	150	1						
Person 2	Pike	253	19	0.01	1.7	119.27	79.46	342	222
	Bass	218	4	0.75	2.0				
	Perch (marine)	181	2	0.13	0.59				

Source: Taken from NMFS (1978).

The above discussions apply to a small proportion of the population at higher risk. Most of the population (99.89%) is subject to exposure to lower levels, less than 0.43 ug/kg/day or 30 ug for a 70-kg person. U.S. EPA (1979) reported that for an average consumption of fish (17 g/day), 3.0 ug/day of mercury would be consumed. This exposure assumed a consumption of 17 g fish/day, 75% of which was tuna (containing 0.2 ug/g mercury). The remaining 25% of the diet consisted of other fish containing 0.1 ug/g of mercury.

Seafood is not the only source of mercury in the diet, although other foods appear to have lower concentrations. Peyton et al. (1975) reported that intake of total mercury would range from 5.3 ug/day to 14.6 ug/day for a standard diet and a range of mercury concentrations in food. The meat, poultry, and fish component of this diet contributed 2.9-8.4 ug/day. The same authors reported a maximum value from the literature of 22.62 ug/day for this group of foods.

The source of methylmercury in the diet, however, is primarily seafood, although other foods also contribute to the total mercury intake (NRC 1978). There is, however, some controversy over how much of the mercury in seafood is methylmercury. Table 22 shows the range of reported results. Cox et al. (1979) point out that the ratio of methylmercury to total mercury is highly dependent on the size of the fish They found no methylmercury in a 23-cm sample, but 0.55 mg/kg in the 35-cm sample.

Thus, without actual measurement of mercury in diets, there seems to be no basis for estimating doses of methylmercury. Both U.S. EPA (1979) and NRC (1978) have assumed that the total mercury intake in fish is in the form of methylmercury. This assumption will certainly provide a worst case estimate of risk.

3. Inhalation

Levels of mercury in air have been discussed in Chapter IV. A, with a wide range of total mercury concentrations reported. The form of mercury in air is generally elemental mercury vapor (NRC 1978) and the inhalation exposure route is expected to contribute little to the body burden of methylmercury. Table 23 shows the inhalation exposure estimates utilized here. The results show that most exposure routes are insignificant compared with the estimated intake through food. Only laboratory or dental office exposures would be in a similar range, and these are occupational exposures, that are subject to the threshold limit of 0.05 mg Hg/m^3. More typical exposures of persons periodically visiting these areas would be considerably lower.

4. Dermal Absorption

Though dermal absorption of mercury ions or compounds in solution may be an important exposure route in certain occupational settings (WHO 1976), it is probably not significant for the general population due to the low concentration of mercury ion or methylmercury usually encountered in natural waters.

TABLE 22. METHYLMERCURY CONTENT OF FISH

Location	Methylmercury (% of Total Mercury)	Source
Swedish	>90	Suzuki *et al*. (1973), as cited in Grieg and Krzynowek (1979)
Japan, Italy, France, Holland	25-30	Vi (1971), as cited in Krenkel (1973)
Tennessee, U.S.	<50	Krenkel *et al*. (1972), as cited in Krenkel (1973)
Maine	~ 100	Rivers *et al*. (1972), as cited in Krenkel (1973)
Freshwater	58	Cox *et al*. (1979)

Blue marlin was the exception - <25%.

TABLE 23. INHALATION EXPOSURE TO MERCURY

Subpopulation	Concentration in Air (ng/m^3)	Exposure[1] (ng/day)	Source
Outdoors			
rural	5	100	(see Chapter IV)
urban	30 (max.)	600	(see Chapter IV)
near sources (natural or anthropogenic)	150-1,500	3,000-30,000	(see Chapter IV)
Indoors			
general	100-200	2,000-4,000	Battelle (1977)
laboratory	200-10,000	4,000-200,000	Battelle (1977)
dental	10,000-100,000	57,000-570,000[2]	Battelle (1977)

[1] Assumes 20 m^3/day respiratory rate.

[2] Assumes 10 m^3 inhalation/working day, 5-day work week

5. Users of Mercury-Containing Products

The consumer is commonly exposed to products containing mercury, specifically thermometers, batteries, lamps, instruments, and paints. With the exception of paints, these exposures are accidental, and thus difficult to quantify. Certainly, the sub-populations associated with such exposures are small. Consumers may also be exposed to mercury through the use of mercury-containing medical or cosmetic products. The dermal absorption of mercury from these sources is expected to be low, although inhalation can occur. In any case, such exposures would generally be insignificant compared with food.

Two incidents of swallowing camera batteries by children have been described in the literature. When new, these batteries contain approximately 2 g mercuric oxide, which could be lethal if released. Unfortunately, these batteries degrade after ingestion and then may come apart. In one case, the battery was extracted, while in the second case it passed through with the child's stool. In neither case was long-term toxicity observed. However, this type of accident may not be unusual, and the increasing use of these batteries is cause for concern.

6. Overview

The primary route of exposure of humans to mercury is through food, especially seafood, which contains methylmercury. An average intake of total mercury in food has been estimated as 5.4-14.6 ug/day. Average consumption of mercury in seafood (methylmercury) is estimated to be 3.0 ug/day. A small proportion (0.1%) of the population (predominantly fisheaters) is subject to exposures of greater than 30 ug/day. An even smaller subpopulation (<0.01%) may be exposed to intakes of mercury of greater than 100 ug/day.

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CHAPTER VI.

BIOTIC EFFECTS AND EXPOSURE

A. EFFECTS ON BIOTA

1. Introduction

This section presents the available information about the levels of mercury that disrupt the normal behavior and metabolic processes of aquatic and terrestrial organisms. Although mercury has received widespread publicity as an extremely toxic substance, the experimental data for biota are not as extensive as might be expected.

A certain amount of inconsistency in the results of bioassay (even for individual compounds) is to be expected, owing to several factors. Some differences may be attributed to the nature of the bioassay procedure (static versus continuous flow), or the use of calculated versus measured concentrations. In addition, water parameters such as temperature and salinity have been shown to affect the toxicity of mercury to various aquatic organisms. Other factors include the species and developmental stage of the test organisms used. Some species and developmental stages may be more sensitive to mercury than others. Variations in these parameters may yield different experimental results and make comparisons among studies difficult.

2. Freshwater Organisms

a. Chronic and Sublethal Effects

Exposure to low levels of mercury may result in acclimation by aquatic organisms, or in behavioral alterations such as ataxia, inappetance, increased respiration, and reproductive inhibition. Prolonged exposure, even to low concentrations of mercury, may ultimately lead to mortality of sensitive species or otherwise decrease the vigor and diversity of local populations to the point that they are endangered.

Panigrahi and Misra (1978) exposed climbing perch (*Anabas scandens*) to 3 mg/l mercuric nitrate for 36 days. After 5 days, the fish became lethargic and reduced their feeding, but regained pre-test behavior 3 days later. After 28 days, however, 71% of the fish had become blind or exophthalmic, and this observation coincided with a marked loss of weight. After 48 hours of exposure to 3 ug/l mercuric chloride ($HgCl_2$). brook trout (*Salvelinus fontinalis*) had increased cough frequency (an effort by the fish to remove accumulated mucus in the gills) (Drummond *et al*. 1974).

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Drummond *et al*. (1974) reported the same effect in brook trout after 8 days' exposure to 3 ug/l methyl mercuric chloride (CH_3HgCl). Rainbow trout (*Salmo gairdneri*) exhibited a loss of appetite during a 120-day bioassay in 860 ug/l CH_3HGCl, and a loss of nervous control after 269 days in 1,600 ug/l CH_3HGCl (Matida *et al*. 1971). In the same study, the growth of the trout was inhibited in concentrations as low as .04 ug/l during an exposure period of 64 days. The growth of alevin brook trout was also reduced in 0.79 ug/l in a chronic bioassay by Christensen (1975).

The only available information on chronic or sublethal toxicity to freshwater invertebrates was from Biesinger *et al*. (1979). Minimum chronic effects levels for *Daphnia magna* were 0.9 ug/l and <0.01 ug/l for $HgCl_2$ and CH_3HgCl, respectively.

Sublethal effects in algae from exposure to inorganic mercury ($HgCl_2$) have been reported for concentrations ranging from 60 ug/l for mixed algae (Blinn *et al*. 1977) to 2,590 ug/l for *Ankistrodesmus braunil* (Matson *et al*. 1972). The effects observed included retarded growth and inhibited rates of chlorophyll synthesis, respiration, and photosynthesis. Enzyme inhibition was reported in the latter species at a concentration of 1,598 ug/l CH_3HgCl.

b. Acute Effects

Data on the acute toxicity of mercury to freshwater biota are compiled and condensed in Tables 24-26. With respect to intra- and inter-species differences, it should be noted that the LC_{50} (concentrations lethal to 50% of test organisms) values given were derived under a variety of conditions.

Surprisingly few species of freshwater finfish have been bioassayed for their sensitivity to inorganic mercury. On the basis of the limited data, rainbow trout appears to be the most sensitive species.

Of the many organic mercury compounds, the five most frequently used in freshwater bioassays are listed, with LC_{50} values, in Table 25. Although the data are sparse, merthiolate and pyridylmercury acetate appear to be less toxic than methylmercury, phenylmercury acetate, or ethylmercury phosphate.

A number of invertebrates have been tested for sensitivity to inorganic mercury (usually $HgCl_2$). All available data are listed in Table 26; no information was found on the toxicity of organomercurics to invertebrates.

3. Marine Organisms

a. Chronic and Sublethal Effects

Information on sublethal mercury toxicosis in marine finfish is

TABLE 24. ACUTE TOXICITY OF INORGANIC MERCURY TO FRESHWATER FINFISH

Fish Species	LC_{50} (ug/l as Hg++)	Reference
Rainbow trout (*Salmo gairdneri*)	33-903	Wobeser (1973), Hale (1977)
Striped Bass (*Roccus saxatilis*)	90	Rehwoldt *et al*. (1972)
Banded killifish (*Fundulus diaphamus*)	110	Rehwoldt *et al*. (1972)
American eel (*Anguilla rostrata*)	140	Rehwoldt *et al*. (1972)
Carp (*Cyprinus carpio*)	180	Rehwoldt *et al*. (1972)
White perch (*Roccus americanus*)	220	Rehwoldt *et al*. (1972)
Coho salmon (*Oncorhynchus kisutch*)	240	Lorz *et al*. (1978)
Pumpkinseed (*Lepomis gibbosus*)	300	Rehwoldt *et al*. (1972)
Channa gachua	1,400	Hanumante and Kulkarni (1979)

TABLE 25. ACUTE TOXICITIES OF ORGANIC MERCURY COMPOUNDS TO FRESHWATER FINFISH

Fish Species	LC_{50} values (ug/l as Hg++)				
	Methylmercury CH_3 Hg Cl	Ethylmercury Phosphate Et Hg Po_4	Phenylmercury Acetate Ph Hg Ac	Merthiolate	Pyridylmercury Acetate
Rainbow trout (*Salmo gairdneri*)	24-42	43	5.1-1,781	10,505	-
Brown trout (*Salmo trutta*)	-	-	-	26,760	2,954
Brook trout (*Salvelinus fontinalis*)	65-84	-	-	39,910	5,082
Lake trout (*Salvelinus namaycush*)	-	-	-	1,055	3,610
Channel catfish (*Ictalurus punctatus*)	-	50	35-3,750	2,800	-
Bluegill sunfish (*Lepomis macrochirus*)	-	-	-	31,960	7,600
Blue gourami (*Trichogaster trichopterus*)	89.5[1]	-	-	-	-

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[1] Roales and Perlmutter (1974).

Source: See U.S. EPA (1979).

TABLE 26. ACUTE TOXICITY OF INORGANIC MERCURY TO FRESHWATER INVERTEBRATES

Species	LC_{50} (ug/l as Hg++)	Reference
Daphnia magna	5	Biesinger and Christensen (1972)
Scud (*Gammarus* sp.)	10	Rehwoldt *et al*. (1973)
Midge (*Chironomous* sp.)	20	Rehwoldt *et al*. (1973)
Crayfish (*Oronectes limosus*)	50-1,000 (LC_{60})	Dayle *et al*. (1976)
Sludgeworm (*Tubifex tubifex*)	82-100	Brkovic-Popovic and Popovic (1977a)
Rotifer (*Philodina acuticornis*)	518-1,185	Buikema *et al*. (1974)
Bristleworm (*Nais* sp.)	1,000	Rehwoldt *et al*. (1973)
Damsel fly larvae	1,200	Rehwoldt *et al*. (1973)
Caddis fly larvae	1,200-2,000	Rehwoldt *et al*. (1973)
Stonefly (*Acroneuria lycorius*)	2,000	Warnick and Bell (1969)
Mayfly (*Ephemerella subvaria*)	2,000	Warnick and Bell (1969)
Snail (*Amnicola* sp.)	2,100	Rehwoldt *et al*. (1973)
Crayfish (*Procambarus clarki*)	200-20,000[a]	Heit and Fingerman (1977)
Crayfish (*Faxonella clypeata*)	200-20,000[a]	Heit and Fingerman (1977)

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[a] 24-96 hour test

limited to a very few species, and excludes most of the organic mercury compounds. The lowest concentration of mercury resulting in sublethal effects was 10 ug/l $HgCl_2$, causing abnormal development in the mummichog, Fundulus heteroclitus (Weis and Weis 1977a) and decreased respiration in the winter flounder, Pseudopleuronecies americanus (Calabrese et al. 1975).

In a study by Cunningham and Grosch (1978), brine shrimp were exposed in different experiments to $HgCl_2$ and CH_3HgCl. Adult reproductive lifespans were significantly reduced at concentrations of 10 ug/l $HgCl_2$ and 5 ug/l CH_3HgCl. The mean number of broods declined as well in concentrations of 1 ug/l and 10 ug/l $HgCl_2$ and in 1 ug/l CH_3HgCl (no lower concentrations were used). However, 10 ug/l $HgCl_2$ had no effect on the average number of offspring produced in each brood, while 1 ug/l CH_3HgCl significantly reduced the fecundity of the shrimp.

The fiddler crab (Uca pugilator) exhibited an increased metabolic rate when exposed to 1.8 ug/l $HgCl_2$. Sublethal effects in other invertebrates included decreased egg and feces production, reduced shell growth, and inhibition of limb regeneration. A summary of these and other data is given with references in Table 27. [For a more complete review of the literature, see Table 13 in U.S. EPA (1979).]

The lowest effects concentrations for marine plants (including algae, diatoms, and kelp) have been reported by Berland et al. (1976), who observed growth inhibition in 18 species of algae in <5 ug/l to 15 ug/l $HgCl_2$. Other sublethal effects such as abnormal development, decreases in chlorophyll mass, and reduced CO_2 consumption have been observed at higher concentrations. Among the nine organomercuric compounds tested, three used by Harriss et al. (1970) reduced photosynthesis in the diatom (Nitzchia delictissima) at a concentration of 0.1 ug/l. [For more detailed information on the toxicity of mercury to marine plants, see Table 11 in U.S. EPA (1979).]

b. Acute Effects

The mummichog is apparently the only saltwater fish that has been tested for acute mercury toxicosis. Both the highest and lowest LC_{50} values (200 ug/l and 6,800 ug/l) have been reported by Dorfmann (1977). For a number of invertebrate species that have been tested, the LC_{50} concentrations range from 3.6 ug/l to 32,000 ug/l. The most sensitive species was the mysid shrimp, closely followed by the hardshell clam and the Eastern oyster. The available data are compiled in Table 28.

4. Other Studies

The only available information on the impact of mercury on ecosystems is a study by Sigmon et al. (1977) on a freshwater community composed of primary producers, herbivores, and carnivorous midges. A one-year exposure to $HgCl_2$ at concentrations of >0.1 ug/l resulted in reduced algae populations, and numbers and diversity of faunal species. No effect on the midges was apparent.

TABLE 27. SUBLETHAL EFFECTS OF MERCURY ON MARINE FAUNA

Species	Compound	Concentration (ug/l as Hg++)	Test Duration	Effect
Mummichog (*Fundulus heteroclitus*)				
embryo	$HgCl_2$	10-20	72 h.	Some development abnormalities
adult	$HgCl_2$, CH_3HgCl	125	24 h.	Disrupted osmoregulation
adult	$HgCl_2$	1,150	96 h.	Aberrant behavior
Winter flounder (*Pseudopleuronectes americanus*) adult	$HgCl_2$	10	60 d.	Decreased respiration, blood chemistry changes
Fiddler Crab (*Uca pugilator*) zoea	$HgCl_2$	1.8	24 h.	Increased metabolic rate
Copepods (*five genera*)	$HgCl_2$	2-10	10 d.	Decrease in egg and fecal pellet production
Copepod (*Pseudocalanus minutus*)	$HgCl_2$	5	70 d.	Growth inhibition
Barnacle (*Balanus balanoides*) cyprid	$HgCl_2$	10	<2 h.	Substrate attachment inhibition
Eastern oyster (*Crassostrea virginica*) adult	$HgCh_2COOH$	10	15 half days	Reduced shell growth
Pacific oyster (*Crassostrea gigas*) larva	$HgCl_2$	32	24 h.	Abnormal development
Polychaete (*Ctenodilus serratus*)	$HgCl_2$	50	21 d.	Reproduction inhibited
Fiddler crab (*Uca* sp.) adult	CH_3HgCl	300-500	32 d.	No limb regeneration

Source: Table 13 in U.S. EPA (1979).

TABLE 28. ACUTE TOXICITY OF INORGANIC MERCURY TO MARINE ORGANISMS

Fish Species	LC_{50} (ug/l as Hg++)	References
Mummichog (Fundulus heteroclitus)	200-6,800	Dorfman (1977)
Invertebrate Species		
Mysid shrimp (Mysidopsis bahia)	3.6-3.9	Sosnowski et al. (1979a)
Hardshell clam (Mercenaria mercenaria)-embryo	4.8	Calabrese et al. (1977)
Eastern oyster (Crassostrea virginica)-embryo	5.6	Calabrese et al. (1977)
Copepod (Acartia clausi)	10	Gentile et al. (1979)
Grass shrimp (Palaemonetes vulgaris)-larva	10	Shealy and Sandifer (1975)
Copepod (Acartia tonsa)	10-20	Sosnowski and Gentile (1978) Sosnowski et al. (1979b)
Polychaete (Capitella capitata)-larva	14	Reish et al. (1976)
Crab (Carcinus maenus)	14-1,200	Conner (1972) Portmann (1968)
White shrimp (Peneus setiferus)-adult	17	Green et al. (1976)
Lobster (Homarus americanus)	20	Johnson and Gentile (1979)
Polychaete (Neanthes arenaceodentata)	22-100	Reish et al. (1976)
Hermit Crab (Pagarus longicarpus)-adult	50	Eisler and Hennekey (1977)
Starfish (Asterias forbesi)-adult)	60	Eisler and Hennekey (197)
Sandworm (Nereis virens)-adult	70	Eisler and Hennekey (1977)
Copepod (Pseudodiaptomus coronatus)	79	Gentile et al. (1979)
Prawn (Pandalus montagui)	80	Portmann and Wilson (1971)
Bay Scallop (Argopecten irradians)-juvenile	89	Nelson et al. (1976)
Copepod (Eurytemora affinis)	158	Gentile et al. (1979)
Copepod (Tigriopus japonicus)	223	Sosnowski et al. (1979b)
Softshell clam (Mya arenaria)-adult	400	Eisler and Hennekey (1977)
Ambassis safgha	2,800	Portmann and Wilson (1971)
Platicthys flesus	3,300	Portmann and Wilson (1971)
Clam (Rangia cuneata)-adult	5,100	Olson and Harrel (1973)
Mud snail (Nassarius absoletus)-adult	32,000	Eisler and Hennekey (1977)

5. Factors Affecting the Toxicity of Mercury

Several variables in a natural aquatic environment may strongly influence the availability and toxicity of mercury to biota. Among these parameters are temperature and salinity; other important factors that have not been adequately tested are pH and water hardness. Fish size and sex have been studied for their association with sensitivity to mercury. The interaction of mercury with other aqueous chemicals may modify its toxicity either by synergy or inhibition; however, such relationships remain to be studied in detail. Perhaps the most important aspect of mercury affecting its toxicity is its chemical form. Although the data are inconsistent, organic forms appear to be generally more toxic than inorganic mercury.

According to MacLeod and Pessah (1973), "temperature is the most important environmental factor controlling rates of biological process" in aquatic biota. In a bioassay with rainbow trout, they found that increasing the temperature from 5° to 20°C decreased the 96-hour LC_{50} from 400 ug/l to 220 ug/l mercuric chloride. In an experiment with six Hudson River fish species, Rehwoldt *et al.* (1972) determined an acute toxicity range of 370-740 ug/l inorganic mercury at 15°C. When the temperature was increased to 28°C, the range of LC_{50} values decreased to 80-420 ug/l. The same effect has been noted for both freshwater and marine invertebrates. Heit and Fingerman (1977) exposed crayfish (*Faxonella clypeata*) to $HgCl_2$ solutions varying between 10^{-7}M and 4×10^{-6}M, and found that the specimens maintained at 20°C survived in greater numbers than those at 30°C. Jones (1973) has reported substantially higher mortality in two estuarine isopods (*Jaera albifrons* and *J. nordmanni*) with a rise of only 5°C (from 10° to 15°C) in 1 mg/l $HgCl_2$. Similar, but somewhat less pronounced results were also observed with two species of marine sowbugs, *Idotea neglecta* and *I. emarginata*.

The effects of variations in salinity are not as well documented, and consequently are less understood. Jones (1973) exposed four species of isopods (above) to 1.0 mg/l and 0.1 mg/l mercury at salinities ranging from 1% to 100% seawater. All species were more sensitive in the less saline solutions, with the most pronounced change in the two estuarine (*Jaera*) species. The combined effect of decreasing salinity and increasing temperature was particularly lethal, possibly because of changes in the rates of absorption. The only other study on the effects of salinity changes on mercury toxicity (Dorfman 1977) indicated no significant trends for the mummichog.

Two studies on mercury toxicity have reported sex-related differences in sensitivities. In two species of crayfish, *Procambarus clarki* and *Faxonella clypeata*, females exhibited substantially more resistance to mercury than males. For example, 50% of the male *P. clarki* test group exposed to 10^{-6}M $HgCl_2$ died within 72 hours, while all the females survived in good health until the end of the experiment at 30 days (Heit and Fingerman 1977). However, a bioassay with brine shrimp (*Artemia salina*) found that females were "physiologically more stressed" than males after exposure to 1 ug/l and 2 ug/l CH_3HgCl. Any differences that do exist between sexes with regard to mercury sensitivity may be species-specific.

Heit and Fingerman (1977) also found that larger specimens of the two crayfish species tested were more resistant to $HgCl_2$ than smaller specimens. In this respect, the results were similar for both males and females.

Heavy metals and other substances often act together to produce or mitigate toxic effects, although few such interactions have been studied in the case of mercury. Calamari and Marchetti (1973) exposed rainbow trout to mixtures of mercury and surfactants. Each surfactant was tested separately in a mercury-detergent pair. The combinations of mercury and anionic surfactants (ABS and LAS) produced toxic effects that were "more-than-additive," while the mixture of nonylphenol ethoxylate (a non-ionic surfactant) and mercury seemed to produce "less-than-additive" effects.

An antagonistic relationship between methylmercury and copper was observed by Roales and Perlmutter (1974) in a bioassay with the blue gourami (Trichogaster trichopterus). While 90 ug/l copper killed 44% and 90 ug/l CH_3HgCl killed 56% of a test group separately, many fewer mortalities occurred when the two metal solutions were mixed in varying proportions. In solutions of 20% Cu/80% CH_3HgCl and 60% Cu/40% CH_3HgCl, all the fish survived the 96-hour exposure period. The authors suggested that the less toxic copper protected the fish from the effects of methylmercury, but no mechanism was hypothesized.

An interaction between mercury and selenium in the natural environment has also been observed. Beijer and Jernelöv (1978) report that these two metals coaccumulate in all marine organisms that have been investigated. They hypothesize that this phenomenon occurs in normal homeostatic regulation, and that the mercury helps the organism retain essential levels of selenium. The authors note that experiments in animals have shown that selenium compounds exert a "protective effect" and decrease the toxic action of organic and inorganic mercury. However, this Hg-Se relationship may have deleterious effects, as it results in increased retention of mercury by the organism which may, in turn, lead to a higher mercury body burden in the individual and an increased rate of biomagnification in the food chain.

It should be noted, however, that the presence of selenium in the tissues is not necessarily a fail-safe protection for all organisms. Harbor seals found along the Netherlands coast exhibited definite signs of mercury toxicosis despite a strong correlation of mercury and selenium in their livers (Koeman et al. 1973).

Although the toxicity data discussed previously in this chapter are subject to various interpretations, organic compounds of mercury are generally considered to be more toxic than inorganic forms. In one comparative study available, Cunningham and Grosch (1978) concluded that methylmercury produced sublethal and lethal effects in brine shrimp at lower concentrations than mercuric chloride. Boney et al (1959) compared the effects of different organomercurics on the red alga, *Plumaria elegans*, and found that toxicity increased with an increase in the number of carbon atoms in the side chain. For example, n = C_3H_7HgCl was found to be approximately four times as toxic to *P. elegans* as CH_3HgCl. Nonalkyl forms such as phenyl- and diphenylmercuries also appear to be substantially more toxic than inorganic mercury. This order of relative toxicity for organomercurials may be reversed in mammals, including man, where methylmercury is very toxic and preliminary data suggest that toxicity of other alkylmercurials decreases with increasing alkyl chain length. Phenyl and diphenylmercury seem even less toxic in mammals, possible due to rapid conversion to inorganic forms in the blood (WHO 1976).

6. Terrestrial Biota

a. Animals

In a 12-month study of the effects of methylmercury dicyandiamide, Heinz (1974) exposed mallard ducks (*Anas platyrhynchos*) continuously to 0.5 mg/kg Hg in the diet. At the end of the study, no health effects were apparent in the adult ducks. Reproductive effects included the production of smaller eggs than controls, and ducks fed 3 mg/kg Hg had only 46.5% as many 1-week-old ducklings as the controls due to hatch failure and mortality. The offspring of the ducks fed 0.5 mg/kg Hg had an increased growth rate compared with the controls and the 3 mg/kg group. No eggshell thinning was observed. The calculated LC_{50} for 10-day-old mallard ducklings over 8 days was reported at 60 mg/kg methylmercury dicyandiamide (Hill, unpublished data). Heinz and Locke (1976) observed that mallard ducklings did not die when ingesting 3 mg/kg methylmercury in their diets, whereas ducklings (fed a clear diet) whose parents were fed 3 mg/kg Hg perished within 3 to 6 days.

A similar experiment on black ducks (*Anas rubripes*) was conducted by Finley and Stendell (1978). Adults were fed 3 mg/kg methylmercury dicyandiamide for 28 weeks in two consecutive breeding seasons. The only apparent effect in adults was hyperactivity in several individuals, which suggested possible mercury poisoning. Again, reduced hatching and higher duckling mortality was found in the test group compared with the control group. However, a slight improvement in reproduction was noted during the second year and residues in eggs, embryos and ducklings of that year were lower than those of earlier offspring. The form of the mercury in these tissues was not determined. Thus, the possibility exists that the hens were better able to metabolize the mercury during the second year. Partial demethylation of the mercury by the hens could account for the improved reproduction and lower embryo residues observed. The authors noted that biotransformation of mercury has been shown to occur in the rat (Norseth and Clarkson 1970) and in guinea pigs (Iverson and Hierlihy 1974).

Studies cited by Heinz (1974) reported mortality in goshawks fed chicken livers containing 13 mg/kg Hg (Borg et al. 1970).

b. Plants

The only information on mercury toxicosis in plants available for this report was a study by Lipsey (1975) on maize seedlings (*Zea mays*). Significant amounts of mercury were absorbed and translocated by the seedlings germinating in CH_3HgOH. Root growth was inhibited when the roots contained >10 mg/kg Hg, while shoot growth was reduced at 0.6 mg/kg.

7. Conclusions

The lowest concentration at which effects were observed in an aquatic organism was <0.01 ug/l CH_3HgCl, a chronic effects value for *Daphnia magna*. Growth was inhibited in rainbow trout at CH_3HgCl concentrations as low as 0.04 ug/l.

Adverse effects on reproduction occurred in brine shrimp at CH_3HgCl concentrations of 1 ug/l. In marine finfish, sublethal effects were observed in 10 ug/l $HgCl_2$ and CH_3HgCl in the mummichog, and in 10 ug/l $HgCl_2$ in the winter flounder. The minimum effects concentration for a marine diatom was 0.1 ug/l for three different organic forms of mercury.

Rainbow trout were again the most sensitive fish in acute bioassays, with LC_{50} values of 5.1 ug/l and 33 ug/l for phenylmercuric acetate and $HgCl_2$, respectively. For all other groups of organisms, only toxicity data for inorganic mercury were found. The LC_{50} for *Daphnia* was 5 ug/l. The mummichog was the only marine fish tested for acute toxicosis, and had a minimum LC_{50} of 200 ug/l. The most sensitive marine invertebrate was apparently the mysid shrimp, with an LC_{50} of 3.6 ug/l.

Studies indicate that the toxicity of mercury increases with increasing water temperature. Some species, particularly estuarine organisms, may be more susceptible to mercury as salinity decreases. It has been suggested that increasing temperature and decreasing salinity act synergistically to increase absorption rates, thus rendering an aquatic organism more susceptible to mercury toxicosis.

According to one study, copper interacts antagonistically with methylmercury, thus effectively reducing the latter's toxicity to aquatic life. Selenium and mercury occur in 1:1 molar ratios in the tissues of all aquatic organisms tested. Apparently selenium mitigates the adverse effects of mercury, but the relationship is not well understood.

Mercury can appear in a variety of compounds, both inorganic and organic, in the environment. The evidence suggests that the organic compounds (particularly alkyl- and phenylmercurics) are more toxic than inorganic forms, and that methylmercury is more ubiquitous than inorganic forms.

Studies of the effects of mercury on terrestrial organisms have been limited. Dietary concentrations of 3 mg/kg CH_3HgCl produced adverse reproductive effects in mallards and black ducks; oral doses of 13 mg/kg and 60 mg/kg were lethal to goshawks and ducklings, respectively. Residues of 0.6 mg/kg and 10 mg/kg in maize seedlings resulted in growth inhibition in the shoots and roots, respectively.

B. EXPOSURE TO BIOTA

1. Introduction

The previous section shows that mercury, especially organic forms, can be toxic to biota. However, as discussed in Chapter IV. A, organic forms of mercury are not predominant in the environment, although levels of organic mercury can be high in the tissues of aquatic organisms. Chapter IV also discussed the routes of exposure to organic mercury and generally concluded that bioaccumulated organic mercury originates from consumption of lower trophic organisms containing these forms, as well as through the rapid accumulation and relatively slow clearance of methylmercury formed in the sediment.

Thus, the implications for aquatic exposure and risk are unclear. Although aquatic organisms are apparently more susceptible to the organic forms of mercury, these forms are rarely found at high levels in natural waters. Aquatic organisms can accumulate methylmercury, although the levels of accumulation have not been specifically correlated with effects on aquatic organisms as they have for humans.

Therefore, although differing by on the order of one to two orders of magnitude for some species, the toxicities of organic versus inorganic forms of mercury were not distinguished in the exposure analysis. The lowest reported effects concentrations, regardless of the form of mercury involved were combined to provide a conservative reference point for use in evaluation of ambient concentrations of total mercury.

2. Monitoring Data for Aquatic Systems

Information on the levels of mercury in the environment is readily available from numerous sources but ambient levels reported are often quite close to the detection limits of analytical methods used during the 1970s. As a consequence, caution is needed when attempting to compare observed levels. In the analysis of aquatic exposure, STORET provided the most comprehensive and internally consistent set of data, and so was the main source used. The main problem with these data was that they do not distinguish between the inorganic and organic fraction of the total. Both organic and inorganic toxicity data for the more sensitive species were used to determine "threshold" mercury concentrations that might be harmful. On the basis of the data summarized in Table 29, mean and maximum values for total mercury of >0.5 ug/l and >10.0, respectively, were chosen for use in the exposure analysis. Concentrations of 0.5 ug/l mercury were selected as an approximate level above which chronic and sublethal toxicosis might appear in sensitive aquatic biota. Since concentrations exceeding 10.0 ug/l are lethal for a number of species under laboratory conditions, this was considered a potential fish kill level. The U.S. EPA (1980) has set the ambient water quality criterion to protect freshwater aquatic life at 0.20 ug/l (total recoverable mercury) as a 24 hr. average, and 4.1 ug/l as a maximum.

TABLE 29. LOWEST MERCURY CONCENTRATIONS HAVING TOXIC EFFECTS ON AQUATIC ORGANISMS

Form of Mercury	Lowest Reported Effect Level (ug/1)			
	Freshwater Invertebrate	Freshwater Fish	Marine Invertebrate	Marine Fish
Inorganic	0.9^{a} (*Daphnia magna*)	[illegible]^{b} (*Salvelinus fontinalis*)	5^{b} (*Pseudocalunus minutus*)	10^{b} (*Fundulus heteroclitus*)
	5^{c} (*Daphnia magna*)	33.0^{b} (*Salmo gairdneri*)	3.6^{c} (*Mysidopsis bahia*)	200^{c} (*Fundulus heteroclitus*)
Organic	0.1^{a} (*Daphnia magna*)	0.04^{b} (*Salmo gairdneri*)	1.2^{a} (*Mysidopsis bahia*)	125^{a} (*Fundulus heteroclitus*)
		5.1^{b} (Salmo gairdneri	150^{c} (*Gammarus duebeni*)	

[a] Chronic value

[b] Sublethal effect

[c] Acute value (LC_{50})

Source: Tables 24-28.

TABLE 30. MINOR RIVER BASINS WITH MEAN TOTAL MERCURY LEVELS EXCEEDING 0.5 ug/l AND/OR MAXIMUM MERCURY LEVELS EXCEEDING 10.0 ug/l, 1979

Basin		Mercury Level (ug/l)		
		Mean	Maximum	No. Samples
1-32	Middle Hudson R.	0.6	-	35
1-33	Lower Hudson - N.Y. Metropolitan Area	1.1	33.0	546
1-34	New Jersey Coast	1.0	-	305
2-3	Delaware R., Zone 1	1.2	-	174
2-5	Delaware R., Schuykill	1.8	25.0	85
2-6	Delaware R., Zone 2	1.6	-	67
2-7	Delaware R., Zone 3	1.4	-	76
2-8	Delaware R., Zone 4	0.8	-	396
2-12	Susquehanna R.	0.7	-	26
3-7	Yadkin - Pee Dee - Lower Pee Dee Rivers	0.8	-	536
3-24	Tampa Bay Area	1.8	34.0	78
3-32	Choctawhatchee R.	0.6	-	9
5-2	Monongahela R.	-	18.0	181
5-6	Hocking R.	0.6	-	24
5-10	Scioto R.	0.7	-	63
5-21	Ohio R., Main stem, minor tributaries	0.7	-	187
7-19	Meramec R.	1.0	-	1
7-22	Mississippi R. - Cape Girardeau Area	2.2	40.0	19
9-4	S. Central Missouri R.	-	20.0	265
9-10	S. Platte R.	0.6	-	45
10-3	Verdigris R.	0.6	12.0	102
10-5	White R.	0.6	11.0	202
10-9	Arkansas R. - Tulsa to Van Buren	0.6	-	105
10-14	Washita R.	0.7	-	30
10-15	Upper Red R. - Above Denison	0.6	-	72
10-21	Lower Mississippi R. - Natchez to Gulf	-	20.0	147
12-5	Colorado R.	1.5	-	35
12-6	Guadelupe Lavaca & San Antonio Basin	1.0	-	22
13-2	Clark Fork - Pend Oreille R.	1.0	-	25
13-3	Spokane R.	0.6	-	483
13-7	Central Snake R.	0.6	-	336
13-8	Middle and Lower Snake R.	0.7	50.0	204
14-3	San Francisco Bay Region	0.7	-	37
14-5	Santa Clara R.	2.4	-	32
14-6	Los Angeles R.	2.7	-	415
14-7	Santa Ana R.	3.2	-	143

Source: U.S. EPA, STORET (1979).

The highest mean levels of mercury in surface water in 1978-1979 occurred mainly in the North Atlantic, Ohio River, South Central Lower Mississippi River, Pacific Northwest, and California basins (see Table 30). The maximum values (> 10 ug/l) appear to reflect isolated events rather than ambient conditions, and are distributed throughout the country.

STORET data for mercury levels in ambient water between 1970 and 1979 indicate generally decreasing levels over this period (see Chapter IV). However, the significance of the changes is uncertain, for example, 1970 may have had unusually high concentrations of mercury, thus distorting any real trend. In order to determine more accurately the trend of aqueous mercury levels, it would be necessary to analyze the data for previous years as well. Data for sediment and fish tissue levels were insufficient to permit a trend analysis. Numerous events of exposure of fish to mercury in the environment have been reported. Incidents of high accumulation levels (exceeding FDA guidelines) are reported occasionally. A discussion of mercury levels and sources of contamination is contained in Chapter IV as well as the Appendix. No fish kills attributed to mercury have been reported in the MDSD Fish Kill Incident files in the last decade.

3. Factors Affecting Aquatic Exposure to Mercury

Certain environmental conditions increase the availability of sediment-bound mercury to aquatic organisms. Rates of mercury methylation in sediment have been observed to increase when a removal mechanism is present. In undisturbed stream beds, methylmercury is generally released very slowly. It is more likely to be released in sediment that is turned over and has greater contact with water, such as in fast flowing streams with bed rolling, in systems supporting active benthic macrofaunal populations, and during spring floods or after rainfall events. Laboratory studies indicate that the half-life of mercury in undisturbed sediment is 6-20 years. However, under natural conditions the half-life is shorter; a half-life of 1-3 years was estimated in sediment in various sections of the Ottawa River (ORPG 1979).

The physical removal of mercury from a local aquatic system occurs via: (1) sediment transport; and (2) desorption from sediment to water and subsequent water transport, with the latter process more significant (ORPG 1979). Chemical variables, such as pH and the composition of the sediment. influence the rate and degree of desorption of mercury from sediment, and thus directly control the availability of mercury for uptake. (Discussion of the adsorption process can be found in Chapter V. B.).

4. Exposure of Terrestrial Organisms

Existing data on the exposure of biota to mercury indicate that terrestrial organisms rarely encounter levels greater than natural background concentrations. With the recent severe reduction in the use of mercury as a fungicide in grain seed, the significance of a major pathway to food chains has declined. However, there are cases of elevated mercury residues in various plant and animal specimens taken near chlor-alkali and pulp/paper mills, and other industrial plants. Moreover, piscivorous birds and mammals often accumulate very high levels of mercury. These species have the greatest potential of all wildlife for exposure to and accumulation of mercury. With their position at the top of the food pyramid, they serve as the most sensitive indicators of environmental contamination by many toxicants.

Many studies on mercury residues in fish-eating mammals and birds are available; however, few have attempted to correlate residues with ambient water levels or proximity to anthropogenic sources of mercury. It is likely that populations near industrial works in which mercury is used are exposed to elevated concentrations of mercury. On the other hand, since the species of concern are highly mobile, their exposure may be intermittent and perhaps insignificant in some cases.

5. Conclusions

The methylated form of mercury is most significant to aquatic organisms in regard to toxicity and bioaccumulation. On the other hand, total mercury is most commonly monitored. At this time it is not possible to estimate organic concentrations from total mercury concentrations for more than a few species. Therefore, except for the discussion of exposure pathways, the exposure analysis is focussed on total mercury levels.

Both direct absorption from water (gill uptake) and ingestion in food appear to be important uptake pathways for aquatic organisms. Although methylmercury concentrations in water are usually below detection limits, they can be great enough to lead to high methylmercury residues in biotic tissue.

Major river basins of the U.S. with the highest concentrations of total mercury in the water column (in 1979) were the North Atlantic, Ohio River, South Central Lower Mississippi River, Pacific Northwest and California regions.[1] Incidents of maximum concentrations exceeding 10 ug/1 were distributed throughout the U.S. and not obviously associated with any particular region or industry.

Reported incidents of aquatic exposure--primarily resulting in significant tissue levels rather than in fatalities--have been associated with the following industries: chlor-alkali plants, pulp and paper mills, and waste disposal ponds from abandoned mines. However, since the reporting of incidents represents only a portion of actual occurrences, it is likely that other sources identified in Chapter III represent potential sources of exposure for aquatic organisms.

[1] Selected on the basis of containing three or more minor river basins with mean Hg concentrations >0.5 ug/1.

The use of mercury in grain seed treatment has been considerably reduced in the last decade, thus obviating one of the more important paths for the entry of mercury into food chains. However, certain industrial and mining operations produce emissions that can increase local mercury concentrations to potentially toxic levels. Tissue samples from animals collected near such sources reveal mercury residues well above the levels in control specimens.

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CHAPTER VII.

RISK CONSIDERATIONS

A. RISKS TO HUMANS

1. Introduction

Previous chapters have described the production and use of mercury and its fate in the environment. It is useful to review the points made previously in order to identify the sources of exposure. Mercury is used primarily in the production of chlorine, in paint manufacture, in the production of instruments, and in the production of electrical equipment, especially batteries. The relative importance of the sources of mercury releases to the environment varies in different regions of the country. In general, sources associated with population centers include losses to air from fossil fuel combustion, incineration of industrial and municipal waste, and from application of paints containing mercury. Losses to the aquatic environment associated with population centers include urban runoff losses from paint and dental applications, and industrial discharges. In some regions, the major sources are copper, zinc and lead mining and smelting. chlor-alkali manufacture. and natural sources, including degassing from the earth and erosion of soils containing mercury.

The monitoring data discussed previously showed that mercury levels in air and soil of urban areas are consistently higher than background levels, and this finding suggests that the sources identified above contribute to exposure of persons in urban areas. Land is the environmental medium which receives the majority of mercury-containing industrial and municipal wastes. Though mercury releases are not expected from properly operated disposal sites, mercury movement from improperly operated sites can be rapid.

The purpose of this section is to compare exposure pathways to humans as described previously in Chapter V with exposure levels at which effects may occur (also described in Chapter V). Consideration of these two elements will aid in the identification of subpopulations that may be exposed to different levels of mercury in its various forms.

2. Major Exposure Routes and Effects Levels

Table 31 summarizes the estimated exposure levels for the major exposure routes. As can be seen, mercury in food represents the largest single source of exposure for the largest number of people. Drinking water and air appear to contribute relatively little to exposure of the general population, although inhalation can be an important exposure route in certain situations, such as near natural or anthropogenic sources.

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TABLE 31. ESTIMATED EXPOSURE OF HUMANS TO MERCURY

Route	Exposure (ug/day)		Form	Subpopulation	Assumptions
	Intake	Absorbed[1]			
Drinking water	<1	<0.1	inorganic	large	Conc. <0.5 ug/l, consumption of 2 l
	4	<0.4	inorganic	very small	Conc. 2 ug/l, consumption of 2 l
Food					
Seafood	3.0	3.0	largely methylmercury	average	Average value of .220 ug/g Hg in tuna, 75% of fish consumption tuna, .100 ug/g in other fish, 17 g/day fish consumption.
	>30	>30	largely methylmercury	pop. 0.1-0.2%	Based on survey at measured concentration and actual consumption.
	80	80	largely methylmercury	<0.0089%	Based on survey at measured concentrations and actual consumption. 0.05-ug/g action limit.
	>100	>100	largely methylmercury	<0.0089%	Based on survey at measured concentration and actual consumption. No action limit.
Total diet	5.3-14.6	2.8-7.9	total mercury	large	Range of conc. in food items, FDA standard diet.
	2.5-7.1	2.5-7.1	methylmercury	large	Range of conc. in food items, FDA standard diet. 85% of mercury in meat, poultry and fish assumed to be methyl.

TABLE 31. ESTIMATED EXPOSURE OF HUMANS TO MERCURY (Continued)

	Exposure (ug/day)				
Route	Intake	Absorbed[1]	Form	Subpopulation	Assumptions
Inhalation					
Outdoors					
rural	0.1	0.08	mercury vapor	large	5 ng/m^3 in air, respiratory rate of 20 m^3/day.
urban	0.6	0.5	mercury vapor	large	30 ng/m^3 in air max.
near sources	3-30	2.4-24	mercury vapor	small	150-1500 ng/m^3 in air.
Indoors					
general	2-4	1.6-3.2	mercury vapor	large	100-200 ng/m^3 in air.
laboratory	4-200	3.2-1.60	mercury vapor	small	200-10,000 ng/m^3 in air.
dental	57-570	40-460	mercury vapor	small	10,000-100,000 ng/m^3 in air; 10 m^3 inhalation, working day, 5-day work week.

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[1] A 10% absorption of ingested inorganic mercury and 100% of methylmercury was assumed. An 80% absorption of inhaled mercury was assumed.

Source: Chapter V.

Table 32 summarizes the lowest observed effect levels, no observed effect levels and "tolerable" or "acceptable daily intake" for mercury as discussed in Chapter V. Of concern are the neurological disturbances and fetal brain damage occurring at relatively low mercury levels in man. Attempts have been made to correlate these blood levels with doses that are also shown in the table. The lowest reported effect levels are based on epidemiologic data and thus represent only obvious effects occurring in the population. Other, more subtle effects may result from lower levels of mercury exposure. The "tolerable" level of 0.43 ug/kg/day was estimated by use of several different methods as described in Chapter V.

Other adverse effects that may be of concern include chromosomal damage, teratogenic effects, and reduction in male fertility. Although these effects have been observed in animals, or in human cells *in vitro*, the significance of these findings to human health effects is unknown.

The following sections will consider the general population and various subpopulations and their exposures to mercury. The exposure levels for these groups will be compared with the "threshold" level believed to result in neurologic disturbances, since at present this is the only effect for which an associated dosage has been estimated. Exposure to fetuses will also be discussed.

3. Risk Considerations for the General Population

Table 33 summarizes estimates of daily absorbed exposure levels for the general population. It is apparent that food is the primary source of methylmercury for this large group. The "acceptable" intake of 0.43 ug/kg/day as discussed in Chapter V is for methylmercury, and thus can only be compared directly with exposure levels of methylmercury. For the FDA standard diet and maximum residues in food, an exposure of 0.1 ug/kg/day can be estimated. There is no direct evidence at this time to indicate that effects would be observed in a population receiving this level of exposure. Statistical analysis of data from several poisoning incidents suggests that the long-term methylmercury intake which produces the earliest symptons in about 5% of the adult population could be 3-7 ug/kg/day (see Chapter V), so any effects associated with this maximum exposure level from food (0.1 ug/kg/day) would probably only be observed in a very small percentage of the total population.

Levels of exposure of the "average" person to mercury through inhalation can approach those from food in some situations. However, mercury in the atmosphere is usually in the form of a vapor, aerosol, or particulate, and these forms do not necessarily have the same dose-response relationship as the forms commonly found in food. At this time it is not possible to establish an acceptable level for inhalation exposures. As shown in Table 32, exposure to concentrations of 0.015 mg/m^3 on a continuous basis may result in effects such as loss of appetite or insomnia. This level is far above the normal range of atmospheric mercury concentrations found in urban or rural areas, though levels near sources (natural or anthropogenic) can approach this effects level and may be of concern.

TABLE 32. ADVERSE EFFECTS OF MERCURY ON MAMMALS

Adverse Effects	Species	Lowest Reported Effect Level	No Apparent Effect Level
Chromosomal damage *in vitro*	Man	13 ng/g erythrocytes (methylmercury)	------[1]
Fetal brain damage	Man	~ 400 ng/g methylmercury in maternal blood ~ 186 mg/kg methylmerucry in maternal hair	------
Neurological disturbances	Man	$\geq$ 200 ng/g blood (methylmercury) = body burden 28-42 mg/kg, dose 3-7 ug/kg/day	20 ng/g blood (methylmercury) 0.43 ug/kg/day estimated dose
Teratogenicity	Mouse	2.5 mg methylmercuric chloride Hg/kg maternal body weight	------
Carcinogenicity	Mouse	------	5000 ng/ml methylmercury drinking water x 70 days; 1000 ng/ml for life
Reduction of male fertility	Mouse	1 mg/kg methylmercury(ip)	------
Appetite loss, insomnia (Hg vapor)	Man	0.06 mg/m^3 mercury vapor (workplace) 0.015 mg/m^3 ambient, estimated	------
Minimum lethal dose	Man	1-4 g $HgCl_2$	------

[1] Effect level unknown.

Source: Chapter V.

TABLE 33. ESTIMATED EXPOSURE OF THE GENERAL POPULATION TO MERCURY

Exposure Medium	Absorbed Dose		Form
	ug/day	ug/kg/day	
Drinking Water	<0.1	0.001	inorganic mercury
Food	2.8-7.9 (2.5-7.1)	0.04-0.11 (0.04-0.10)	total mercury methylmercury
Air	1.2-2.5[1]	(0.02-0.04)	mercury vapor

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[1] Combines indoor and outdoor exposures with the assumption that 75% of time is spent indoors.

Source: Table 31.

Occupational exposures may be high in dental offices, as well as in other occupational settings. However, exposures of occasional visitors to these locations would be much lower.

As discussed previously in Chapter V, humans can be exposed to mercury from silver-mercury dental fillings. Though this type of exposure has been observed, it has not been quantified in a way which permits evaluation of risk to this large exposed population at the present time.

4. Risk Considerations for Subpopulations

a. Fisheaters

Table 31 shows that 0.1% to 0.2% of the U.S. population may receive more than 30 ug/70 kg person/day or 0.43 ug/kg/day methylmercury in seafood. The data on consumption patterns and mercury concentrations in various fish species used in this estimate are based on two separate surveys. In the consumption survey, the actual concentrations in the seafood consumed were not measured. Although the FDA action level of 1.0 ug/g mercury is in effect for fish, 100% compliance is not expected. In addition, in setting the guideline the FDA assumed a consumption level of 30 g fish per day, and a small percentage of the population probably consumes more than this. Effects of mercury exposure would not necessarily be observed in the 0.2% of the population, since the sensitivity to mercury varies.

Geographic areas in which levels of mercury in seafood are high can be identified to some extent. They have been discussed in Chapter V and in the Appendix. The localized areas in which consumption of contaminated fish is high are not easily identified. A survey conducted by the National Marine Fisheries Service showed that consumers of large amounts of seafood were concentrated in the North and Mid-Atlantic states, the Southeast, the Great Lakes states, and in Texas, California and Oregon (see Chapter V). Thus, it appears that a small portion of the population from these areas, although they are large, may be at risk due to consumption of mercury.

The types of fish eaten by persons with a mercury intake exceeding 0.43 ug/kg/day include both freshwater and saltwater species (see Table 34). Possible sources of mercury for freshwater species containing high mercury concentrations include natural sources, chloralkali plants, mining, copper smelters, and power plants. Electric lamp, battery, instrument, and paint manufacturers may also be sources in local areas. Sources contributing to large bodies of water, like the Great Lakes, would be numerous. The sources mentioned above would probably contribute as well as runoff, agricultural use of fertilizer and pesticides, emissions from paint applications and POTWs.

Mercury levels in saltwater species appear to be largely due to "natural bakcground" levels rather than a specific source. Accumulation of high mercury levels are probably due to the large size of some marine species.

An examination of the areas of the country where freshwater fisheries have been restricted due to mercury showed that the cause of contamiantion included natural sources, abandoned chlor-alkali plants, and an abandoned gold mine. However, in many cases the sources could not be identified.

b. Fetuses

Fetal brain damage has been shown to result from mercury exposure to the mother, as discussed in Chapter V, 4, c. Minimum effects levels have not yet been established, but clinical evidence of fetal brain damage has been observed in a study involving 20 mother-infant pairs when peak maternal hair mercury concentration rose above 100 mg/kg (estimated to be equivalent to 400 ng/ml blood concentration). In a separate incident, severe fetal brain damage was correlated with a peak maternal hair concentration of 186 mg/kg. However, it has been estimated that the earliest effects of mercury toxicity would be observable in the most sensitive adult population at blood levels in the range of 200-500 ng/g. Taken in conjunction with the fact that neurological effects have not always been obvious in mothers of infants with clinical evidence of brain damage from mercury, there is some basis for inferring that minimum effects levels for fetal brain damage may be less than or equal to 200 ng/ml maternal blood concentrations.

Since only 30 mother-infant pairs were involved in these studies on fetal brain damage, it was not necessarily a representative sample. To deal with this uncertainty, some state governments which have closed or otherwise limited fisheries have recommended that pregnant women not consume certain species of fish.

c. Children

The risk to children due to mercury exposure may be of concern due to the indications of higher susceptibility of this subpopulation. Because relatively little is known regarding the dose-response relationship for mercury in children, detailed exposure analyses were not included for them. However, the risk to children should be at least as great as that for adults.

d. Users of Mercury-Containing Products

The accidental exposure of consumers to mercury does occur in a very small subpopulation. Although this exposure is unquantifiable, it is probably low relative to food exposure.

The swallowing of camera batteries by children is one such route since these batteries are becoming more widely used in the home. The corrosion of the casing may expose the child to potentially lethal dose.

TABLE 34. FISH SPECIES CONSUMED BY SEAFOOD EATERS WITH MERCURY INTAKE EXCEEDING 0.43 ug/kg/day

Species	No. persons eating (of 47)
tuna (light)	24
bass (sea and striped)	16
pike	15
flounder	12
perch - marine	10
mackerel (other than jack)	8
halibut	4
haddock	4
swordfish	2

Note: Other species such as crappie, sunfish, trout, shrimp, lobster, salmon, etc., were consumed, but infrequently.

Source: See Hall *et al*. (1978), discussed in Chapter V.

B. RISKS TO BIOTA

The exposure analysis for biota (Chapter VI) suggested that mercury levels have gradually decreased in the major river basins of the U.S. since 1970. Monitoring data on levels in bottom sediment and fish residues, however, are inadequate to permit a similar trend analysis for these media.

STORET data from 1978 to 1979 indicate that the highest mean mercury concentrations were in the North Atlantic, Ohio River, South Central Lower Mississippi, Pacific Northwest, and California basins. In all of these regions, total aqueous mercury levels often exceeded 0.5 ug/l, a concentration which has sublethal effects in several species in the laboratory. In addition, there are a few instances of maximum mercury concentrations exceeding 10.0 ug/l, which is an acute toxicity level for some aquatic organisms. Such incidents are not concentrated in any one area, but appear to have occurred across the country.

The main difficulty in interpreting these data is an uncertainty with regard to the chemical form of the mercury at any given location. The data as reported in STORET do not distinguish between organic and inorganic mercury, but rather describe total mercury levels in water, sediment, and fish tissues. The toxicity information presented in Chapter VI suggest that methylated mercury is usually more toxic than inorganic mercury. A conservative approach would be to assume that all mercury is in this, its most toxic form, despite the fact that most aqueous mercury is inorganic. Even with this assumption, however, the risk to aquatic organisms cannot be quantified on the basis of the available data. Consequently, the minor basins listed in Table 30 (Chapter VI) do not necessarily reflect aquatic populations at risk; at best, they represent regions where the greatest hazards may exist.

Most marine fish are probably protected from mercury toxicosis as a result of the relatively high selenium levels in ambient seawater. In inland waters, however, selenium is normally a much less significant component, and so, consequently, freshwater fish are probably more susceptible to mercury. Toxicity data are not extensive enough to allow confident identification of the more sensitive species. The rainbow trout and the daphnids seem to be among the most sensitive groups; however, these are also among the most frequently bioassayed species. One group of special interest is crayfish, for some of which the LC_{50}s for mercury are several orders of magnitude below those for other freshwater species.

Most terrestrial organisms do not appear to be at risk, except perhaps in the vicinity of certain anthropogenic sources. Elevated mercury residues have been found in plant and animal specimens taken near chlor-alkali chemical plants, although no toxic responses were noted. Piscivorous mammals and birds may be exposed to more mercury than other animals because of their position of the top of the food pyramid.

APPENDIX A

NOTES TO TABLE 1

1. Bureau of Mines (1978).

2. Bureau of Mines (1976).

3. SRI (1979).

4. The U.S. Bureau of Mines (1976) reports that 607 flasks of mercury (at 76 lbs each) were consumed in the production of fungicides and bactericides. This amount corresponds to approximately 20.9 kkg. SRI (1979) found similar results.

5. The amount of mercury deposited in industrial stockpiles is taken to be the difference between the total for production and imports (2428 kkg) and the total for known consumption (2253 kkg) for 1976.

6. Van Horn (1975). Emission volumes have been rounded to one decimal.

7. Assumptions regarding the relative amounts of mercury used in the three principal subcategories of electric apparatus (tubes/switches, lamps, and batteries) were made by Versar, Inc., after reviewing the literature (Van Horn 1975, Battelle 1977, Versar, Inc. 1976b, United Technology 1975) and contacting the battery industry (Personal communication, Irwin Frankel, Mallory Battery Co., Tarreytown, NY, 1976). Factors relating to fraction of mercury lost, and emission factors were obtained from Van Horn (1975):

Elect. App.	% of Total	Consumption (kkg)	Fraction Lost(%)	Distribution to Environmental Media(%)			Hg lost (kkg)		
				Air	H_2O	Landfill	Air	H_2O	Landfill
Tubes/ Switches	10	94.8	0.025	-	-	1.00	-	-	2.4
Lamps	10	94.8	0.04	.05	-	.95	0.2	-	3.6
Batteries	80	758.4	0.005	.05	.02	.95	0.2	0.1	3.6
TOTAL	100	948					0.4	0.1	9.6

948.0 kkg consumed in manufacturing
- 10.1 kkg lost to environment during manufacturing
937.9 kkg in products, generally having extended lives

Data are not available concerning the amount of mercury discharged to POTWs, but some small part of the aquatic discharge is assumed to go to POTWs.

8. Since all mercury purchased by the chlor-alkali industry is eventually lost by a variety of routes, and since no additional mercury-cell plants have come into existence in recent years, it can be assumed that the equivalent of the purchased mercury ends up in the environment or in the product. Reasonably accurate data are available for 1975 regarding the amount of mercury that is discharged to air, in plant effluents and caustic soda. Most of the remainder ends up in landfills, or in land-locked slurry ponds, evaporation ponds, or is recycled to deep brine wells, all of which are also considered to be the land compartment.

In 1975, data from 16 of the 27 chlor-alkali plants showed that the average loss of mercury to the air was 1.73 kg/day/plant (including losses to byproduct hydrogen) (Versar, Inc. 1976a). For an assumed operating factor of 98%, losses to air were:

$$1.73 \text{ kg/day} \times 27 \text{ plants} \times 365 \text{ day/yr} \times .98 = 16.7 \text{ kkg/yr}$$

When the above is adjusted to account for the somewhat greater use of mercury by the industry in 1976, the total is computed as:

$$16.7 \text{ kkg/yr} \times \frac{10.06 \times 10^6 \text{ tons of } Cl_2 \text{ in } 1976}{9.1 \times 10^6 \text{ tons of } Cl_2 \text{ in } 1975} = 18.5 \text{ kkg}$$

Similarly, loss of mercury to caustic product at 16 plants was 0.27 kg/day/plant (Versar, Inc. 1976a)

$$.27 \text{ kg/day} \times 27 \text{ plants} \times 365 \text{ days/yr} \times .98 \times \frac{10.6 \times 10^6 \text{ tons}}{9.1 \times 10^6 \text{ tons}} \times \frac{1 \text{ kkg}}{1000 \text{ kg}}$$

$$= 2.9 \text{ kkg/yr in NaOH product in 1976}$$

Mercury losses via plant effluents for 23 plants were 0.046 kg/day/plant, therefore

$$27 \text{ plants} \times 0.046 \text{ kg/day} \times 365 \text{ days/yr} \times 0.98 \times \frac{10.06 \times 10^6 \text{ tons}}{9.1 \times 10^6 \text{ tons}} \times \frac{1 \text{ kkg}}{1000 \text{ kg}}$$

$$= .5 \text{ kkg/yr to plant effluent in 1976}$$

According to Jacobs (1979), the average mercury discharge for 12 plants was 10^{-4} kg/kkg of Cl_2. The amount of chlorine produced by this process is reported to be 2,750,000 kkg (in 1977). In the total subcategory for the electrolytic preparation of chlorine and caustic soda, there are 77 plants, 72 of which discharge directly, and five that discharge to POTWs. There are 27 plants in the subcategory that use the mercury-cell process. Hence:

$$\text{Aquatic Discharge} = 10^{-4} \text{ kg/kkg} \times 2{,}750{,}000 \text{ kkg} \times 1 \text{ kkg/1000 kg}$$
$$= 0.3 \text{ kkg}$$

Because the total aquatic discharge of mercury is small, and the number of plants discharging to POTWs from the entire subcategory is small, it is assumed that all of the mercury is discharged directly and that the discharge to POTWs is negligible.

Since the total consumption of mercury by this industry in 1976 was 553 kkg, and losses to air, water, and product can be accounted for, it can be assumed that all the difference goes into landfills (or sludge ponds, evaporative ponds, and brine wells). This difference, in 1976, was:

553 kkg - 18.5 kkg - 2.9 kkg - 0.3 kkg = 531.3 kkg to land

9. Approximately 270 kkg of mercury were used in paint manufacture during 1976, largely in the form of mildewcides, such as phenyl mercuric acetate and phenyl mercuric oleate or succinate. The portion of mercury lost from paint manufacture is 0.1% (0.27 kkg) (Van Horn 1975). If 5% of losses are assumed to be to air, 5% to land, and 90% to water (Van Horn 1975), then the amounts of mercury lost to the media are:

Air: 0.05 x 0.27 kkg = 0.015 kkg = ~0 kkg
Water: 0.9 x 0.27 kkg = 0.24 kkg = ~0.2 kkg
Land: 0.05 x 0.27 kkg = 0.015 kkg = ~0 kkg

However, during the screening and verification sampling and analysis programs for the paint manufacturing industry, 22 plants were sampled. The mercury concentrations in treated effluent ranged from 0 ug/l to 2900 ug/l, with an average concentration of 580 ug/l (U.S. EPA 1979c). The total daily water discharge for direct dischargers is 25,000 gal/day and for indirect (POTW) dischargers it is 750,000 gal/day (Burns and Roe 1979). For 250 operating days per year:

Direct discharge = 580 ug/l x 25,000 gal/day x 3.785 l/gal
x 250 days/yr x 10^{-12} kkg/ug
= 0.01 kkg
= ~0 kkg

Discharge to POTWs = 580 ug/l x 750,000 gal/day x 3.785 l/gal
x 250 days/yr x 10^{-12} kkg/ug
= 0.4 kkg

Since these estimates for the aquatic discharge and discharge to POTWs are based on actual sampling data, they are given in Table 1.

The amount of mercury that is actually used in paint products is the amount consumed by the industry (270 kkg) less the amount lost during production (0.4 kkg), or 269.6 kkg.

After application of paint, 65% of the mercury is volatilized to the air (U.S. EPA 1973); thus of the 269.6 kkg of mercury used in paints, 175.2 kkg is lost to the air. If the remaining 94.4 kkg is assumed to be evenly distributed among landfills, land fallout (due to paint flaking), runoff, and the air (as a result of incineration), then the total environmental distribution from paint use is:

Air:	23.6 + 175.2 = 198.8 kkg	
Water (runoff):	23.6 kkg	
Landfill:	23.6 kkg	47.2 kkg
Land Fallout:	23.6 kkg	

There are no data by which to partition the mercury between direct aquatic sinks and POTWs; therefore, the entire amount has been put under the aquatic discharge heading.

10. Approximately 175 kkg of mercury were used in the manufacture of industrial and control instruments. According to Van Horn (1975), the emission factor for the industry is 1% of the amount consumed, and all of this is assumed to be in the form of solid waste:

Solid waste discharge = 0.01 x 175 kkg = 1.75 kkg = 1.8 kkg

During the use of these instruments, all of the mercury is expected to either enter the environment or to be recycled.

Total mercury available = 175 kkg - 1.8 kkg = 173.2 kkg

The following emissions distribution was reported by Van Horn (1975) as follows:

Air:	4%	6.9 kkg
Water:	-	-
Land	56%	97.0 kkg
Recycled:	40%	69.3 kkg
TOTAL		173.2 kkg

11. For dental work, about 68.6 kkg of mercury was used in 1976, and of this approximately 22% was lost to water and 2% was lost to air during application while the remainder was in the dental work of the patient (Van Horn 1975).

If one assumes that about one-third of the amalgam in humans ends up in landfills (burial) when the patient dies and that about two-thirds has to be replaced during the lifetime of the patient, then of the 76% that is not lost to water and air during application, the two-thirds that is replaced amounts to 51% of all mercury used in fillings, and it too is lost to water. Thus, the total environmental distribution of mercury lost to the environment is:

73% to water (i.e., 22% + 51%)
2% to air
25% to land.

Of the 73% in wastewater, two-thirds are assumed to go to POTWs. Thus the annual mercury burden to the various environmental media is as follows:

Air:	1.4 kkg
Water:	16.5 kkg
POTWs:	33.6 kkg
Land:	17.1 kkg

12. Approximately 43.6 kkg of mercury was used in catalyst manufacture in 1976, mostly in catalysts for vinyl chloride and manufacture of vat dyes. About 0.25% (0.1 kkg) was lost to air and 0.5% (0.2 kkg) to wastewater during manufacture (Van Horn 1975). There are no data available from which to determine the amount of mercury that is discharged directly or to POTWs; therefore, the entire amount estimated is listed as an aquatic discharge. Data are not available by which to estimate the solid wastes from this industry; however, it is not expected to be significant.

    It is assumed, but has not been verified that the remainder (43.3 kkg) was disposed of in landfills, when the spent catalysts are discarded.

13. Approximately 21 kkg of mercury are used in agricultural pesticides (Bureau of Mines 1976). Negligible amounts are lost during manufacture. When the pesticides are used, it is estimated that 15% (3.1 kkg) is lost in runoff and reaches the aquatic environment and 85% (17.9 kkg) goes to land (Van Horn 1975).

14. In 1976, approximately 20.5 kkg of mercury was used in various laboratories, including college, high school, hospital, and independent research laboratories. The following distribution was estimated by Van Horn (1975).

Air:	10%	2.1 kkg
Water:	26%	5.3 kkg
Landfill:	7%	1.4 kkg
Recycle:	57%	11.7 kkg
	TOTAL	20.5 kkg

    Data are not available by which to determine the amount of mercury that is discharged to POTWs or discharged directly to the aquatic environment. Since most of the laboratories are located in cities, it is assumed that the major portion of this discharge would be to POTWs.

15. About 2.1 kkg of mercury is used in the manufacture of pharmaceuticals, and only about 0.001 to 0.002 kkg is lost during manufacture; this is considered to be negligible (U.S. EPA 1976c).

During the consumer use of mercury-bearing pharmaceuticals, it is estimated that 90% is lost to water (i.e., 60% goes to POTWs plus 30% to direct discharge), and 10% goes to landfills. Thus the annual releases to the various compartments total:

Water	0.63 kkg
POTWs:	1.26 kkg
Landfills:	0.21 kkg

16. Batteries manufactured from mercury are contained in a metal case with a plastic seal. Therefore, little if any mercury is likely to be released during use. However, in the United States (unlike Japan and some European countries), there is no organized recycling program, and substantially all batteries enter the municipal solid waste stream (Arthur D. Little, Inc., estimate). Though the mercury cells contain the highest concentration of mercury, a larger total quantity of mercury is consumed in the manufacture of alkaline-manganese dry cells, and nearly all dry cells contain some mercury.

In general, the batteries containing the highest concentrations of mercury are also the ones that are built most ruggedly, and are, therefore, most likely to survive waste handling processes such as compaction. The seal is the weakest point in the battery and will probably be oxidized (over time) and release the contained mercury.

If one assumes a relatively benign environment at the landfill site, one can speculate that mercury cells, and to some extent alkaline-manganese dry cells, may act as leak-proof containers of mercury for 20-50 years (and perhaps longer). If corrosive conditions prevail in the landfill, then the lifetime would be shorter. (Arthur D. Little, Inc., estimate). Dry cells in that portion of municipal solid waste that is incinerated would presumably rupture, and allow most of the mercury to escape to the atmosphere.

For the purpose of this materials balance, it is assumed that 758.4 kkg of mercury was consumed in batteries in 1976 (80% of the total in this category), and that 15% of municipal solid waste is incinerated, with the remaining 85% placed in landfills. Thus, 637.8 kkg of mercury will go to land, and 112.6 kkg will go to the atmosphere from the disposal of batteries containing mercury.

The remaining sources of mercury to the environment from electrical apparatus are described in the text.

17. Mercury emissions arise from the combustion of three fossil fuels: coal, oil, and natural gas. Most steam electric power generating plants can be classified as predominantly coal burning, oil burning, or gas burning. Power generation and fuel consumption in 1975 was as follows (National Coal Association 1976):

208,632 MW from 483,588,000 tons of coal in 381 coal plants
66,362 MW from 456,032,000 bbls oil in 533 oil plants
72,326 MW from $295{,}915 \times 10^{7}$ ft^3 natural gas in 422 gas plants

During 1976, new plants were brought in with total additional power ratings of (National Coal Association 1976):

11,976 MW for coal
4,410 MW for oil
1,436 MW for gas

The fuel consumption for 1976 was therefore:

$$\frac{483{,}588{,}000 \times (208{,}632 + 11{,}976)}{208{,}632} = 511.35 \times 10^{6} \text{ tons coal}$$

$$\frac{456{,}032{,}000 \times (66{,}762 + 4{,}410)}{66{,}762} = 486.2 \times 10^{6} \text{ bbl. oil}$$

$$\frac{(295{,}915 \times 10^{7}) \times (72{,}326 + 1{,}436)}{72{,}326} = 3.018 \times 10^{12} \text{ ft}^{3} \text{ natural gas}$$

Mercury contents of these fuels are as follows:

0.2 mg/kg for coal (U.S. EPA 1973)
0.066 mg/kg for distillate oil (Van Horn 1975)
0.13 mg/kg for residual oil (U.S. EPA 1973)
0.04 mg/kg in natural gas (Van Horn 1975)

In 1972, the total fuel oil used in the U.S. was $1{,}066 \times 10^{6}$ bbl. of distillate and 973,707,000 bbl. of residual or 53% and 47%, respectively (Bureau of Mines 1972). The amount of each used for electric power generation is not known, but if the same proportions are assumed to have been used for power generation, both in 1972 and 1976, the average mercury concentration in fuel oil is:

$$(.066)\ (.53) + (.13)\ (.47) = .096 \text{ mg/kg}$$

The mercury emitted by the combination of fossil fuels is:

$$(511.35 \times 10^{6} \text{ tons coal})\ (.2 \text{ mg/kg})\ (.907 \text{ kkg/ton}) = 92.8 \text{ kkg Hg from coal}$$

$$(486.2 \times 10^{6} \text{ bbl})\ (7.82 \text{ lb/gal})\ (0.096 \text{ mg/kg})\ (0.907/2000) = 6.95 \text{ kkg Hg from oil}$$

$$(3.018 \times 10^{12} \text{ ft}^{3})\ (76.4 \text{ lb/1000 ft}^{3})\ (0.554)\ (0.04 \text{ mg/kg}\ (0.907) = 2.32 \text{ kkg Hg from natural gas}$$

(Note: 0.554 = sp. gr. of methane, and methane constitutes >98% of natural gas.)

When coal is burned: 90% of mercury goes to flue gas
9.4% goes to land (ash)
0.6% goes to water (ash pond overflows)

Mercury in environmental media as a result of fuel combustion for steam electric power generation is, therefore (Van Horn 1975):

		Distribution to Environmental Media (%)			Hg to Media (kkg)			
Fuel	Hg in fuel(kkg)	Air	Water	Land	Air	Water	Land	Total
Coal	92.8	.90	.006	.094	83.5	0.56	8.7	
Oil	6.95	.999	-	.001	7.0	-	0.006	
Gas	2.32	.999	-	.001	2.3	-	0.002	
TOTAL	102.1				92.5	0.56	8.7[1]	102.1

[1] Quantities less than (<) 0.1 kkg/year are disregarded.

18. Coal use was as follows according to the Bureau of Mines (1979):

665,000,000 tons bituminous coal mined in 1976
1,150,000 tons bituminous coal imported
(60,000,000) tons bituminous coal exported
6,200,000 tons anthracite coal mined
612,350,000 tons = total used in U.S.
- 511,350,000 tons coal for steam electric power generation
101,000,000 tons = total coal combusted, other than in steam electric power generation

Hg discharged from coal = (101×10^6) (0.2 mg/kg) (.907 kkg/ton) = 18.3 kkg Hg

Natural gas use in the U.S. in 1976 was distributed roughly as follows (Bureau of Mines 1979):

$19{,}500 \times 10^9$ ft^3 = total natural gas used
- $3{,}018 \times 10^9$ ft^3 = natural gas used for steam electric power generation
$16{,}500 \times 10^9$ ft^3 = natural gas used for other than steam electric power generation

Hg discharged from gas =
$(16{,}500 \times 10^9 \text{ ft}^3)$ $(76.4 \text{ lb}/1000 \text{ ft}^3)$ (0.554 sp. gr.) (0.04 mg/kg) (0.907/2000) = 12.7 kkg Hg.

Oil use in the U.S. in 1976 was approximately as follows (National Coal Association 1976):

4810×10^{6} bbls (42 gal/bbl) = total domestic oil demand (90% used for fuel)

436.10×10^{6} bbl used for steam electric power generation

Thus: $(.9)(4810 \times 10^{6}) - 486.2 \times 10^{6} = 3842.8 \times 10^{6}$ bbl oil was used for other than steam electric power generation

Hg discharged from oil=
$(3842.8 \times 10^{6}$ bbl) (42 gal/bbl) (7.82 lb/gal) (0.096 ppm)
(0.907/2000) = 54.9 kkg

Mercury in Environmental Media (Van Horn 1975)

Fuel	Hg in Fuel (kkg)	Distribution to Environmental Media (%) Air	Water	Land	Hg to media (kkg) Air	Water	Land
Coal	18.3	.90	.006	.094	16.5	0.1	1.7
Oil	54.9	.999	-	.001	54.8	-	.1
Gas	12.7	.999	-	.001	12.7	-	-
TOTAL	85.9				84.0	0.1	1.8

19. According to SRI (1979), 35% of sewage sludge is incinerated, 15% is dumped in the ocean, 25% is spread on land as agricultural fertilizer, and 25% is placed in landfills. SRI also states that the total amount of mercury in sewage sludge is between 0.26 kkg and 203.2 kkg. Thus, the partition of mercury in sewage sludge between the various environmental media is:

Air (incineration):	<0.13 to 71.1 kkg (assuming most of the Hg is volatilized during incineration and that the rest is landfilled)
Land (fertilizer):	0.09 to 50.8 kkg
Land (landfilling):	0.09 to 50.8 kkg

Intermediate values were chosen for Table 1 and Figure 1.

20. According to the 1979 Development Document for Ore Mining (Calspan 1979), the only producing mine in the U.S. - the McDermitt Mine - has a zero effluent discharge, except, of course, for non-process wastewaters, which are assumed to be negligibly contaminated. Furthermore, since most mining operations are in the western states where rainfall is rare, runoff from tailing and other mine refuse piles is also assumed to be negligible.

21. According to the 1979 Development Document for Nonferrous Metals (U.S. EPA 1979a), primary and secondary production plants for mercury have a zero effluent discharge. (See Note 20)

22. Only limited screening and verification data are available for the Steam Electric Power Generation Industry (U.S. EPA 1979c). Mercury was detected in the effluents at relatively low concentrations in all but one subcategory. Five of the seven subcategories have a significant discharge. The following data were available (U.S. EPA 1979c):

Subcategory	No. Plants Sampled	No. Plants in the Industry	Average Flow (MGD)	Average Conc. (ug/1	Discharge (kkg)
Cooling Tower Blowdown	9	250	2.4	3.9	3.2
Fly Ash Transport	10	312	2.0	0.1	0.1
Metal Cleaning Wastes	6	750	3.3×10^{-5}	21,286	0.7
Low Volume Wastes	3	1068	0.28	0.6	0.3
Air Pollution Control System Blowdown	5	10	0.98	16	0.2
				TOTAL	4.5 kkg

(Note: Discharge = (No. of plants) (Avg. Flow) (Avg. Conc.)
(365 days/yr) (3.785 l/gal) (10^{-12} kkg/ug)

There is low confidence in this estimate because only a few plants were sampled (usually less than 15 per subcategory), and the numbers were extrapolated to subcategories with a large number of plants and large flow volume per plant.

23. Mercury was detected in the effluents of the Timber Products Processing Industry during the verification sampling and analysis program (U.S. EPA 1979c). Mercury is discharged in significant amounts from two subcategories - Hardboard S1S and Insulation Board (Thermochemical). The following data were available (U.S. EPA 1979a):

Industry Subcategory	No. Plants in Subcategory	No. Plants Sampled	Avg. Flow (gal/day/plant)	Avg. Conc. (ug/1)	Discharge (kkg)
Hardboard S1S	8	1	8,236,600	4	0.3
Insulation Board (Thermochemical	4	2	13,803,190	5.5	0.3
				TOTAL	0.6

(Note: Discharge = (No. of plants) (Avg. Flow) (Avg. Conc.) (250 days/yr) (3.785 l/gal) (10^{-12} kkg/ug)

24. Mercury was detected in the effluents of petroleum refineries during the verification sampling and analysis program (U.S. EPA 1979c). The average concentration was 0.16 ug/l and average flow per plant was 3.3×10^6 gal/day. There are 182 direct dischargers and 48 indirect (POTW) dischargers (U.S. EPA 1979b). Thus:

$$\text{Direct discharge} = (182 \text{ plants}) (0.16 \text{ ug/l}) (3.3 \times 10^6 \text{ gal/day}) (365 \text{ days/yr}) \times (10^{-12} \text{ kkg/ug}) (3.785 \text{ l/gal}) = 0.1 \text{ kkg}$$

The mercury discharge to POTWs is considered to be negligible since it is on the order of only one quarter of the amount discharged directly.

25. The amount of mercury discharged from POTWs was taken to be the average of three separate sets of data.

Data Set #1 - Municipal treatment plants generate 0.017 kkg of sludge per person per year, and 1.6×10^8 people are serviced by POTWs (ASMA 1976). In a 1976 study of the sludges from 16 cities, Furr _et al_. (1976) report a mercury concentration of 8.6 mg/kg. The annual discharge of mercury in sludge is, therefore:

$$(0.017 \text{ kkg/person}) (1.6 \times 10^8 \text{ people}) (8.6 \times 10^{-6}) = 23.4 \text{ kkg}$$

It is assumed that 65% of all mercury entering sewage plants is removed in sludge before wastewaters are discharged (Davis and Jacknow 1975). Thus, the total amount of mercury discharged both as sludge and in POTW effluents is:

$$23.4 \text{ kkg}/0.65 = 36.0 \text{ kkg}$$

The amount of mercury discharged in the effluent is the difference between the total and that remaining in the sludge:

$$36.0 \text{ kkg} - 23.4 \text{ kkg} = 12.6 \text{ kkg}$$

Data Set #2 - A 1973 survey of 56 sewage treatment facilities indicated that the average effluent concentration of mercury was <0.003 ug/l while a 1977 survey of discharge monitoring reports from 12 treatment plants also indicated a mercury concentration of <.003 ug/l. The total effluent flow for POTWs is estimated to be 22,670 MGD (U.S. EPA 1976b). The estimated annual discharge is:

$$(<0.003 \times 10^{-3} \text{gm/l}) (22.67 \times 10^9 \text{ gal/day}) (3.785 \text{ l/gal}) (365 \text{ days/yr}) (10^{-6} \text{ kkg/gm}) = <94.0 \text{ kkg}$$

This amount is considered to be the maximum amount of mercury discharged from treatment plants.

Data Set #3 - It has been reported that 19.9 kkg of mercury is released annually to public waters from POTWs (University of Illinois 1978).

The total reported in Table 1 is the average of the results of the above three calculations:

(12.6 kkg + 94.0 kkg + 19.9 kkg)/3 = 42.2 kkg

26. Mercury was detected in the effluents of the Auto and Other Laundries Industry during the verification sampling and analysis program (U.S. EPA 1979c). Almost all of the plants in this industry discharge to POTWs. Therefore, the direct aquatic discharge for this industry is considered to be negligible. The following data were available for this industry (U.S. EPA 1979c).

Industry Subcategory	No. plants	Avg. Flow (gal/day)	Avg. Conc. (ug/1)	Discharge (kkg)
Industrial Laundries	1,020	75,000	2.3	0.2
Power Laundries	3,094	10,000	2.3	0.1
Car Washes	77,693	5,000	0.8	0.3
Linen Supply	1,314	60,000	1.6	0.1
			TOTAL	0.7 kkg

27. According to SRI (1979), 190.5 kkg of mercury is added to the environmental burden each year as a result of the mercury contained in fertilizers. Some of this mercury may enter the aquatic environment by means of runoff, but the amount is not known and there is no basis for an estimate.

APPENDIX B

STATUS OF RESTRICTIONS ON COMMERCIAL AND SPORT FISHING DUE TO MERCURY CONTAMINATION

In June of 1977, an inventory was taken of states that have, since 1970, closed sport or commercial fisheries and/or issued health warnings concerning the consequences of eating fish or other seafood contaminated with mercury (NAS 1978). Since June of 1977, two main factors have led to the lifting of the bans/warnings in many states: the FDA has raised the action level for mercury in fish tissue from 0.50 ug/g to 1.00 ug/g, and many industries that were discharging mercury-containing effluent directly have either stopped discharging altogether or currently treat their wastes.

Three levels of restrictions are addressed in the following summary:

(1) states with current closures, restrictions or advisories;

(2) states in which consumption warnings are in effect;

(3) states in which prior closures, restrictions or advisories have been rescinded.

In the description that follows, changes that have occurred since the 1977 survey are reported; if no change occurred, the 1977 status is presented. For states with restriction categories (1) and (2) above, the appropriate state health or environmental official was contacted. The rationale for limiting this effort was that those areas with the most serious existing or past mercury pollution problems warranted the most attention and states that fall into category (3) were not contacted.

STATE	CURRENT STATUS
Alabama*	A 1970 restriction on commercial fishing in the Tombigbee, Tensaw, and Mobile Rivers and their respective tributaries,

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* States that have rescinded closures of sport and/or commercial fisheries or health warnings issued since 1970.

** States in which health warnings are in effect about the consequences of eating mercury-contaminated fish or other seafood from selected water-courses in the state.

*** States where sport or commercial fisheries are currently closed and health warnings are in effect.

STATES	CURRENT STATUS
	as well as the waters of Upper Mobile Bay, was lifted on July 7, 1972. However, all of the Pickwick Reservoir in Alabama was closed to commercial fisheries between 1970 and 1975. There are no consumption advisories in effect (Samuel Spencer; Department of Conservation and Natural Resources, State of Alabama; personal communication, April 1980).
California**	A warning to eat only one meal per week of striped bass and catfish from the Sacramento-San Joaquin Delta and San Francisco Bay area was issued by the State Department of Health and is still in effect. In addition, in 1972, warnings were issued by the Santa Clara County Park and Recreation District that fish (largemouth bass, sunfish, catfish, and rainbow trout) taken from Calero, Almaden, and Guadalupe reservoirs may contain high levels of mercury and should not be eaten (NAS 1978).
Georgia*	In 1970 the Savannah River and New Savannah Dam on Highway 17, as well as the Brunswick Estuary, were closed to sport fishing. The Brunswick Estuary was also closed to commercial fishing. All restrictions and closures for the Brunswick Estuary were removed on October 19, 1970, and were removed for the Savannah River in September 1972 (NAS 1978).
Idaho*	No state restrictions or fishery closures are currently in effect. Conditional warnings (no person should eat more than 1/2 lb of fish per week; and pregnant women, infants, and children should not eat any fish taken from American Falls Reservoir) were issued by the State Health Department for selected species of fish in the American Falls Reservoir (January 1971 and 1972), Hells Canyon Dam, Jordan Creek, and other reservoirs on the Snake River (January 1971), but have since been removed. Sources of mercury to these water bodies are thought to be industrial or agricultural (American Falls Reservoir and Hells Canyon Dam), an abandoned gold mine (Jordan Creek), and natural sources (the Snake River) (Stacy Beghards; Fisheries Division, Department of Fish and Game, State of Idaho; personal communication, April 1980).
Illinois**	Before 1970, there were sport or commerical fishery closures and no health warning advisories to fishermen or the public about the consequences of eating mercury-contaminated fish. In 1970, however, certain species of fish taken from three reservoir lakes (Rend Lake, Cedar Lake, and Lake Shelbyville) exceeded the FDA action level at that time of 0.5 ug/mercury. As a result, the public was warned to limit consumption to no more than 1/2 lb per week of largemouth bass, shorthead redhorse, black buffalo, bullhead, and yellow bullhead from these lakes. The advisory has since been dropped for Cedar and Shelbyville Lakes. No mercury problems have been

STATES	CURRENT STATUS
	identified that affect commercial fisheries. Sources of mercury to the waters of the restricted lakes are unknown; however, it is likely that the mercury is naturally occurring [William Fritz; Department of Conservation (Fisheries), State of Illinois; personal communication, April 1980].
Kentucky*	The health warning and restrictions issued in 1970 for fish taken from the Tennessee River at Calvert, Kentucky, have been relaxed due to a drop in mercury levels. A state-run sampling program involving 30 stations is currently in progress, and the results will be available in early 1981 to verify the levels of mercury in the Tennessee River (Robert Logan; Division of Water Quality, Department of Natural Resources and Environmental Protection, State of Kentucky; personal communication, April 1980).
Louisiana*	In 1970, Louisiana issued a health warning regarding fish taken from the Calcasieu River and stopped the interstate shipment of these fish. All state restrictions were removed in 1975 because the mercury concentrations in fish were below the FDA tolerance level of 0.5 ug/g then in effect (NAS 1978).
Massachusetts*	In 1970, minor fisheries were closed and health warnings were issued for three specific areas. As a result of mercury contamination above the FDA action level of 0.5 ug/g, two shellfish areas were closed in December 1970: Sippican Harbor in Marion and Quisset Harbor in Falmouth. In 1975, portions of these harbors were reopened to shellfishing because mercury levels had declined. Neither area was heavily industrialized, and the source of mercury was believed to be marinas in which mercury-based paint was being used in boat yard work. Also in 1970, a health warning was issued for persons who were engaged in recreational finfishing in the Taunton River. Fish could be taken, but people were advised not to consume those from the northern boundary of the Town of Fall River north to the northern boundary of the Town of Dighton. This warning was the result of an industrial discharge, which has since been terminated. All health warnings have since been withdrawn (John Jonasch; Fisheries Biologist, Division of Water Pollution Control, State of Massachusetts; personal communication, April 1980).

STATES	CURRENT STATUS
Michigan***	On April 15, 1970, sport fishing was banned and health warnings posted on the St. Clair River and Lake St. Clair. Commercial fishing for walleye in Lake Erie was banned on April 29, 1970. On May 20, 1970, the sport fishing restrictions were reduced to "catch and release" status in the St. Clair and Detroit Rivers and Lake St. Clair. In Lakes Erie, Huron (south of Port Sanilac), and St. Clair, sport fishermen could keep all fish except walleye, white bass, and freshwater drum, while commercial fishermen could keep all species except walleye. A public health advisory remains in effect for Lake Superior (lake trout), Lake Michigan (salmon), Lake St. Clair (large and small mouth bass). Sport fishing continues to be restricted in the Detroit and St. Clair Rivers. The sources of mercury in Lake Michigan and Lake Superior are unknown; it is believed that the mercury contamination in the other water bodies is attributable to industrial discharges or runoff (James Forney; Toxic Materials Branch, Fisheries Division, State of Michigan; personal communication, April 1980).
Minnesota**	There have been no closures of sport or commercial fisheries in the State. On December 11, 1970, the Department of Health advised anglers to restric intake of fish from certain waters to once a week due to high mercury levels. Subsequent analyses of fish for mercury resulted in modification of the warning between 1970 and 1976. The following four watercourses were found to contain some fish exceeding the FDA action level of 0.5 ug/g: (1) the St. Louis River below Coloquet, (2) the Upper Mississippi River between Grand Rapids and Brainerd, (3) the Red River along the Dakota border, and (4) Crane Lake near the Canadian border. The latest modification to health advisories occured on May 14, 1976, when the Department of Health advised that fish from Crane Lakes be eaten no more than once a week (Larry Gust; Environmental Health Division, Minnesota Department of Health; personal communication, June 1980).
Mississippi**	On August 1, 1975, the Mississippi portions of Pickwick Lake were reopened to commercial fishing. The Mississippi State Board of Health also issued a warning that pregnant women should restrict consumption of fish from Pickwick Lake to a minimum and that all other persons should limit their normal intake of fish from this lake to not more than two meals per week (Charles Chisholm; Director of Air and Water Pollution Control Commission, State of Mississippi: personal communication; June 1980).

STATE	CURRENT STATUS
New Hampshire*	No state restrictions are in effect. The warnings issued in 1970 for pickerel, yellow perch, and smallmouth bass from the Merrimack and Connecticut Rivers have been removed because public health officials believe that the current creel limits preclude anyone from eating sufficient quantities of fish to be harmful to health (Charles Thoits; Inland and Marine Fisheries Branch, Fish and Game Department, State of New Hampshire; personal communication, April 1980).
New Mexico**	Sport fishery health cautions for the Navajo and Ute Lakes were issued by the Health and Social Services Department (HSSD) in 1970 and are still in effect. The public has been advised not to eat more than 2 lb per week of any species of fish taken from Navajo Lake. If walleye and largemouth bass weighing more than 1.5 lb were taken from Ute Lake, the recommended consumption was to be limited to less than 1 lb per week per adult person, and the recommended consumption of catfish weighing more than 5 lb was limited to 2 lb per week per person. Warnings were also issued against eating large amounts of fish taken from Summer, Elephant Butte, and Caballo Lakes. The HSSD stressed that it was safe to eat the fish provided that the recommended consumption limits were observed. No point sources of mercury contamination have been identified. Local authorities have speculated that a possible source is runoff from abandoned placer mines, which used "quick silver" (David Tague; Bureau of Water Pollution Surveillance, Health and Social Services Department, State of New Mexico; personal communication, April 1980).
New York***	With the exception of fish from three bodies of water, officials have proclaimed that it is safe to eat fish once a week without fear of mercury contamination. Onondaga Lake is closed to fishing. People were advised not to eat lake trout from Lake George or muskellung from the St. Lawrence River, but the warning has been lifted. Pregnant women and infants are advised not to eat any freshwater fish. Some lakes in the Adirondacks have been found to contain borderline concentrations of mercury but no action has been taken in that area. There are no restrictions on commerical fishing. The source of mercury pollution appears to be natural, except in the case of Onandaga Lake where a chlor-alkali plant had a significant daily discharge. Wastes from this plant are now being treated and the condition is expected to improve (Edward Horn; Bureau of Environmental Protection, State of New York; personal communication, April 1980).

STATE	CURRENT STATUS
North Carolina*	For the inland fisheries, the general danger warnings issued in 1970 to fishermen are no longer in effect. No closures or health warnings have been issued for the marine fisheries. However, the FDA ban on swordfish resulted in the closure of a small fishery for this species on the northern coast of this State (Robert Benton; Marine Fisheries, Department of Natural Resources and Community Development, State of North Carolina; personal communication, April 1980).
Ohio*	In 1970 the Lake Erie commercial fishery was closed for all fish except perch. An embargo was placed on white bass and a sport fishery health warning announced. Since then the 1970 restrictions were ruled unconstitutional by the Ohio Supreme Court because "the Division of Wildlife is not and was not responsible for consumer protection." No state restrictions or health warnings are presently in effect (NAS 1978).
Oregon*	There are no commercial fishery closures in effect. However, in 1970 health warnings were issued for rainbow trout, black crappie, suckers, and largemouth bass taken from the Antelope and Owyhee reservoirs and parts of the Willamette River. A curtailed intake of any fish taken from these waters was recommended, particularly for infants and pregnant women. In 1975 a health warning was issued for striped bass (NAS 1978).
Pennsylvania*	In 1970 the Department of Environmental Resources issued an advisory that large predator game fish, such as walleye, drum, smallmouth bass, and white bass, may exceed the FDA action level of 0.5 ug/g mercury, and, therefore, some restriction of the consumption of these fishes by humans may be advisable. At the present time there are no official restrictions on catching game fish, and no health warnings have been issued with respect to eating the species (NAS 1978).
South Carolina**	In 1970 the sport and commercial fisheries were closed on the Savannah River from Augusta, Georgia, to the coast. These restrictions were removed in 1972. In 1972 an advisory was issued that recommended limiting the consumption of fish taken from Lake Jocassee to 1.5 lb of dressed fish per week and eliminating intake by pregnant women. The elevated levels of mercury in fish from Lake Jocassee were the result of natural conditions, such as the slightly higher soil mercury levels in the lake area and, more significantly, the oligotrophic condition of the lake. The advisory is

STATE	CURRENT STATUS
	currently in effect, and the mercury levels are being monitored (J. Luke Hause; Division of Shellfish and Recreational Waters, Department of Water and Natural Resources, State of South Dakota; personal communication, April 1980).
South Dakota**	In 1970, the State reported no closures or advice to fishermen about health hazards associated with eating fish taken from South Dakota waters. Since then, only the Cheyenne Arm of Oahe Reservoir has been posted by the State's health officer. In June 1973, commercial and sport fishermen were warned not to eat more than 1.5 lb of fish from this water per week. This health warning is still in effect. The source of mercury is believed to be runoff from an abandoned gold mine in the Black Hills (James Nelson; Water Quality and Hygiene, Department of Water and Natural Resources, State of South Dakota; personal communication, April 1980).
Tennessee***	In September of 1970, the Tennessee River and Pickwick Lake commercial fisheries were closed, a health warning was issued, and a catch and release policy instituted for sport fishing in these areas. Both the commercial and sport fisheries restrictions were removed for Pickwick Lake and the Tennessee River in August of 1971. A catch and release restriction and health warning imposed on sport fishing in the North Fork Holston River in September 1970 is still in effect and commercial fishing is also not allowed. The source of mercury is runoff from a closed chlor-alkali plant in Virginia (Elmo Lunn; Water Quality Control Division, State of Tennessee; personal communication, April 1980).
Texas**	In 1970 approximately 19,900 acres of Lavaca Bay were closed to commercial oyster harvest because of an accidental spill from a chlor-alkali plant. This was a single occurrence, and any detectable mercury levels today can be attributed to runoff of residual mercury from the spill or from natural sources. In September 1971 the size of the restricted area was reduced from 19,900 acres to 11,000 acres. Currently safety warnings are in effect for only a small area adjacent to the site of the spill. Additionally, the restrictions on the harvesting of oysters were not entirely because of mercury pollution; prior to its reclassification in 1970, Lavaca Bay had approximately 8500 acres that were closed because of sanitary and bacteriologic reasons (Lloyd Crabb; Shellfish Program, Bureau of Environmental Health, State of Texas; personal communication, April 1980).

STATE	CURRENT STATUS
Vermont*	In 1970 Lake Champlain and its tributaries were closed to the commercial harvest of walleye. In addition, an embargo was placed on commercial sales of walleye from Lake Champlain, its tributaries, and Lake Memphremagog, and the embargo is still in effect. On April 25, 1973, the sport fishery danger warnings imposed in 1970 were continued for the consumption of walleye from Lake Champlain, its tributaries, and Lake Memphremagog. After a state sampling program, all other restrictions were lifted in the mid 1970s. Mercury concentrations are attributable to natural sources (Wally McClane; Water Resources Division, State of Vermont; personal communication, April 1980).
Virginia***	In 1970 the sport fishery on the North Fork of the Holston River below Saltville was closed by the Virginia Department of Health due to contaminated runoff from a closed chlor-alkali plant. In 1975 this restriction was relaxed to permit fishing under a catch and release regulation, and the restriction was completely lifted in 1977. A health warning was issued in 1970 and again in 1975 concerning the danger of eating fish taken from these waters; this warning is still in effect. On June 6, 1977, the Virginia Department of Health closed the sport fishery on the South River, the south fork of the Shenandoah River between Waynesboro and the Page County line, which is restricted to a "catch and release" policy. Citizens are warned that fish taken from these waters are unfit for human consumption (Robert Stroube; Bureau of Toxic Substances, Department of Health, State of Virginia; personal communication, April 1980).
West Virginia*	Sport and commercial fisheries in West Virginia are presently not restricted due to mercury pollution. The Ohio River commercial fishery, which was closed on August 29, 1970, was reopened on July 1, 1973. Currently, West Virginia has no health warnings in effect about the consumption of mercury-contaminated fish (NAS 1978).
Wisconsin*	In 1970 a catch and release policy was recommended for the Wisconsin River, along with a health warning that not more than one meal of fish taken from this river should be consumed each week. As of 1977, there were no state restrictions because mercury levels in the Wisconsin River system had dropped below the FDA action level of 0.5 ug/g. Contracts for commercial fishing are now being granted for the Wisconsin River and its impoundments, and warnings on fish consumption limits are no longer being issued (NAS 1978).

REFERENCE

National Academy of Sciences (NAS); An assessment of mercury in the environment; Washington DC: National Research Council; 1978.